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ABSTRACT

This book contains additional items submitted for the record to the hearings dealing with legislation designed to improve public awareness about the health effects of smoking. The materials contain letters from health professionals and organizations concerned with prevention of heart disease, lung disease, dental problems and cancer. Research is highlighted that links smoking to various health problems, along with discussions about the type, amount, and content of cigarette advertising. Statements from those opposed to the proposed legislation are included, citing inconclusive research and the rights of consumers to make an informed decision. (JAC)

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COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT

APPENDIX TO HEARINGS

BEFORE THE

SUBCOMMITTEE ON
HEALTH AND THE ENVIRONMENT

OF THE

COMMITTEE ON
ENERGY AND COMMERCE
HOUSE OF REPRESENTATIVES

NINETY-SEVENTH CONGRESS

SECOND SESSION

ON

H.R. 5653 and H.R. 4957

MARCH 5, 11, AND 12, 1982

Serial No. 97-107

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American Council on Science and Health

Dr. Elizabeth M. Whelan
Executive Director

Dr. David B. Roll
Associate Director

Anne M. Christopher, M.S.
Development Director

December 3, 1981

Honorable Henry A. Waxman
CONGRESS OF THE UNITED STATES
Rayburn House Office Building
Room 2415
Washington D.C. 20515

Dear Congressman Waxman:

Thank you for your recent letter informing me of H.R. 4957. I am writing to lend support to this bill and your efforts to increase the public's understanding of the deleterious health effects of cigarette smoking.

ACSH has frequently been critical of government efforts to warn citizens of the alleged health effects of saccharin, nitrite and other food additives. We object in these cases because there is no adequate data to support the hypotheses that these substances pose a risk to human health.

In the case of cigarette smoking, however, the evidence is overwhelming. I believe it is the correct role of government to provide educational information on health risks to consumers, and let the consumers make their own decisions.

Providing a more specific description of the consequences of smoking may indeed convince some smokers to at least think about quitting. From that point of view, it is a good idea and thus our support of H.R. 4957. However, I hope you and others stress that more education through labeling should be only one part of our plan to convince Americans to give up tobacco smoking. Ultimately, I feel, the answer will be to shift more of the burden of smoking's effect to the smoker. This is already beginning in the private sector with the differential insurance rates for smokers and nonsmokers.

Please call on us if we can help in any way with the passage of your bill.

Sincerely,

Elizabeth M. Whelan
Executive Director

EMW:cem

Please Reply to:
☐ 1995 Broadway 18th Floor New York, NY 10023 Telephone: 212 362 7044
☐ 47 Maple Street Summit, NJ 07901 Telephone: 201 277 0024
☐ 1111 16th Street, N.W. Suite 301 Washington, D.C. 20036 Telephone: 202 659 0978

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December 10, 1981

The Honorable Henry A. Waxman, Chairman
Subcommittee on Health and the Environment
U.S. House of Representatives
Washington, D.C. 20515

Dear Congressman Waxman:

The American Lung Association is pleased to endorse H.R. 4957, the "Comprehensive Smoking Prevention Education Act of 1981". Reduction in the number of Americans who smoke and the approximately 350,000 who die annually as a result, is a major program objective of ALA.

Certainly providing legal authority for the Office of Smoking and Health is an important step toward strengthening the focus on anti-smoking education within the Federal government. Educational efforts by OSH must be continued.

You may be interested in ALA's letter to the Federal Trade Commission wherein we comment on the warning labels suggested by FTC staff that seemed especially effective to us. ALA believes that rotation of warnings is a device well worth trying.

In addition to coordinating efforts of government agencies, the establishment of the Interagency Committee on Smoking and Health will enable outside groups such as ours and others represented on the voluntary National Interagency Council on Smoking and Health to work with the government in a more effective manner than is now the case. A governmental coordinating body should be a significant step in moving forward the campaign against smoking.

It is our hope that legislation to discourage smoking will now progress in Congress and that ALA will be given an opportunity to testify in support of your bill.

Sincerely,

Edmund C. Casey, M.D.

Edmund C. Casey, M.D.
President

cc: Robert V.P. Hutter, M.D., President, American Cancer Society
James A. Schoenberger, M.D., President, American Heart Association

/cc

Founded in 1904, the American Lung Association includes affiliated associations throughout the U.S., and a medical section, the American Thoracic Society

3

american
medical
student
association

p.o. box 131
14650 lee road
chantilly va 22021

703/968-7920

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December 18, 1981

Honorable Henry A. Waxman
Chairman, Subcommittee on Health
and the Environment
Rayburn House Office Building
Washington, D.C. 20515

Dear Mr. Waxman:

The American Medical Student Association (AMSA) would like to express its gratitude and support for your introduction of the Comprehensive Smoking Prevention Education Act of 1981 (HR 4957) on November 12th. We believe that this legislation will result in a significant, cost-effective coordination of federal and private educational activities regarding the adverse effects of smoking on health. Our organization has a long history of concern about the enormous costs to our nation--in terms of human life as well as health resources--that are associated with widespread smoking of cigarettes and other tobacco products.

AMSA is an independent national organization representing over 25,000 students of allopathic and osteopathic medicine at 130 institutions in the United States. It is dedicated to the principle that access to health care is a right, not a privilege Therefore, such access to equally high standards of health care must be provided regardless of economic status. Moreover, AMSA is committed to furthering the orientation of medical practice toward the achievement of health, a "positive, dynamic state of physical, mental, and environmental well being," not merely the treatment of disease.

Consistent with the expressed philosophic goals of promotion of health prevention, AMSA has made significant commitments of effort and organizational resources to smoking prevention education. In our "Preamble, Purposes and Principles," a substantial amount of organizational policy addresses our concern with the issue of tobacco smoking and its adverse effects on health. Included in our "Principles Regarding Quality of Life and the Environment" AMSA:

- SUPPORTS the use of federal tax on cigarettes to fund increased research on the prevention/treatment of cancer and cardiovascular disease and increased disease prevention programs;

- SUPPORTS increased public education programs regarding the health hazards of cigarettes and other tobacco products;

C 8

- •URGES the use of federal, state and local funds for television and radio anti-smoking messages as a major component of the anti-smoking effort, and URGES that an increased federal tax on cigarettes be specifically used to supplement such funds; and
- •OPPOSES the continuation of federal price supports of tobacco crops.

In addition, our organization maintains a national Nutrition and Preventive Medicine Task Force, which performs the function of disseminating information about preventive efforts, such as this bill, to our membership, and a standing Legislative Affairs Committee, which voices our concerns regarding policy issues which affect the health of the American people, such as HR 4957. We also maintain a program by which national organizational funds may be directed to individual chapters for local projects, such as for development of smoking prevention education efforts. From our experience in developing these programs within our organization, it has become obvious that programs of far greater scope are needed, requiring continuous development on a national level, if the public's awareness of the health hazards of smoking is to be maintained.

In this letter we do not need to present any of the biomedical data regarding the adverse health effects of smoking which have led AMSA to inclusion of these statements in our "Principles," or to establish sections addressing the educational and legislative concerns of our membership regarding this issue. It is our belief that the current biomedical data present overwhelming evidence that a significant reduction in the practice of cigarette smoking by our population would result in a dramatic improvement in morbidity and mortality statistics, particularly those reflecting the incidence of some forms of cancer, chronic respiratory diseases, and heart disease. Therefore, we ardently support efforts to increase and coordinate smoking prevention education efforts within the public and private sector, such as providing statutory protection to the Office on Smoking and Health and replacement of the current cigarette health warning, as described in HR 4957.

We would be most happy to provide whatever support you feel would be most appropriate for the successful outcome of this bill. We feel that, as the largest independent medical student organization, AMSA could provide a rather unique aspect of support, reflecting our prespective and concerns as both present and future providers of medical care. We look forward to working with you on this matter of great mutual concern.

Sincerely,

John C. Carl

John C. Carl
Legislative Affairs Coordinator

Californians for Nonsmokers' Rights

(formerly Californians for Smoking & No Smoking Sections)

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Berkeley, CA 94701
(415) 841-3032

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December 21, 1981

Honorable Henry A. Waxman
House Office Building
Washington, D.C. 20515

Dear Congressman Waxman,

On behalf of Californians for Nonsmokers' Rights, I would like to thank you for introducing HR 4957.

Californians for Nonsmokers' Rights is an organization with more than 25,000 contributors that grew from the two recent initiative campaigns on the question of smoking in public places. We are continuing to work on issues relating to nonsmokers' rights in particular and smoking and health in general through the political process at all levels in California.

Since it bears so directly on your bill, I am enclosing, for your information, a copy of a letter I recently sent to the Federal Trade Commission endorsing the conclusions and recommendations in their "Staff Report on the Cigarette Advertising Investigation." I am also enclosing letters from faculty of the U.C. San Francisco and Stanford Medical Schools that attest to the strength of the conclusions in this report.

The only change we suggest in your bill is that the system of rotational warnings be expanded to include one or more warnings to educate the public that smoking harms nonsmokers as well as smokers, such as

WARNING: Your smoke hurts people with heart disease

WARNING: Your smoke hurts nonsmokers

WARNING: Your smoke hurts your children

WARNING: Nonsmokers inhale poisons from your smoke

My letter to the FTC outlines the rationale for these suggestions in more detail.

Although our organization is primarily concerned with local and state-wide legislation in California, we were proud to see that a California Congressman chose to introduce this important bill. I am also writing to the rest of the California Congressional delegation asking that they support your bill. If we can do anything else to help secure its passage, please let me know.

Sincerely yours,

Stanton A Glantz

Stanton A. Glantz, Ph.D.
Treasurer
Associate Professor of Medicine
University of California, San Francisco

Californians for Nonsmokers' Rights

(formerly Californians for Smoking & No Smoking Sections)

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December 4, 1981

Secretary
Federal Trade Commission
Sixth and Pennsylvian Avenues, NW
Washington, D.C. 20580

Re: File 792-3204

Dear Sir:

I am writing you to express Californians for Nonsmokers' Rights' endorsement of the contents and recommendations in the FTC "Staff Report on the Cigarette Advertising Investigation."

Californians for Nonsmokers' Rights is a statewide organization supported by more than 25,000 contributors that is concerned with protecting nonsmokers from the harmful effects of cigarette smoke in particular and issues of smoking and health in general. We have been organized (under a variety of names) since 1978, when we ran the first of two statewide initiative campaigns on smoking in public places.

We have asked professionals from the University of California and Stanford University to review the Report, and the consensus is that it represents the definitive study on the effects and goals of cigarette advertising, the surprising public ignorance of the harmful effects of smoking, and the need for a better system of warning the public about the dangers of smoking.

The finding that the public is not as well informed about the dangers of smoking as one would expect is an extremely important one, since smoking represents the major avoidable cause of disease in the United States. Our experience in running two initiative campaigns on smoking-related issues in California supports the statistical results in the Report. We routinely encountered people who downplayed the medical evidence that smoking is dangerous. At the time we thought we were dealing with unusually poorly informed or recalcitrant individuals; but, as the Report so convincingly demonstrates, we were simply encountering a widespread ignorance concerning the ill effects of smoking.

We found the discussion of cigarette advertising compelling, especially pages 2-15 through 2-24, which dealt with specific efforts by Brown and Williamson to induce young people to start smoking and to keep them smoking despite the fact that smokers realize that their behavior is fundamentally irrational.

The claim of cigarette manufacturers that advertising is not intended to convert nonsmokers to smokers but only to influence brand choice is also belied by recent developments within the industry, including the increasing use of general circulation magazines, in particular women's magazines, for advertising. By its own admission, the industry has targeted women as a vulnerable group. A front-page article in the September 28, 1981, *Advertising Age* titled "Women Top Cigarette Target" quotes Gerald H. Long, President and Chief Executive Officer of R.J. Reynolds Tobacco, as describing the women's market as "probably the largest opportunity for Reynolds." Reproductive risks are increased in women who smoke and lung cancer rates are increasing so dramatically that it will soon become the number one cancer killer among females in the United States, a trend due virtually entirely to cigarette smoking. Yet, the number of pages of magazine advertising devoted to images of attractive, healthy-looking young women in cigarette promotions has increased radically over the past decade. For example, *Better Homes and Gardens*, the fourth ranking magazine in total circulation in the U.S. in 1980, now averages 15 full pages of cigarette ads per monthly issue.

These advertisements present a powerful message associating smoking with vigor, youth, and health, a message that clearly conflicts with the realities of suffering and death that accompany smoking. The present abstract warning message stands little chance of having an impact in this context. In addition, since reading the Report, I have taken note of how the warning is located on the advertisements in a visually isolated place. The layout of the ad always moves your eye away from the warning to a part of the ad that presents smoking as a positive experience. This effect contrasts sharply with the arrow and circle design suggested in the Report that always attracted attention to the important health message it contained.

All the recommendations are sound and should be implemented as soon as possible.

We would suggest, however, that you also include warnings that address the danger of smoking to nonsmokers. We suggest this for two reasons. First, as the enclosed bibliography and the discussion of involuntary smoking in the recent National Academy of Sciences/National Research Council report *Indoor Pollutants* (National Academy Press, 1981) demonstrate, there is a significant body of scientific evidence associating so-called passive smoking with danger to nonsmokers. Second, concern about nonsmokers' rights may be the most effective way to reduce the amount of smoking. As the Tobacco Institute's 1978 Roper Poll observed, the development of the nonsmokers' rights movement is "the most dangerous development to the viability of the tobacco industry that has yet occurred." The Tobacco Institute's recent national advertising campaign to discredit work by Hirayama (published in the *British Medical Journal* in January, 1981), that demonstrated that non-smoking wives of smoking husbands had an increased incidence of lung cancer, is powerful evidence that the tobacco industry is concerned about growing public knowledge that passive smoking is harmful. Therefore, we would like to suggest that you add one or more of the following warnings to the rotational warning system:

WARNING: Your smoke hurts people with heart disease.

WARNING: Your smoke hurts nonsmokers.

WARNING: Your smoke hurts your children.

WARNING: Nonsmokers inhale poisons from your smoke.

We specifically endorse the proposal for rotational warnings; it will provide more information to the public in a way that will be much more likely to be noticed.

If we can provide any additional information, please call on me.

Sincerely yours,

Stanton A Glantz

Stanton A. Glantz, Ph.D.
Treasurer
Associate Professor of Medicine
University of California, San Francisco

SAG/mh

Enclosure

cc: California Congressional Delegation

Stanford
Heart Disease
Prevention Program
School of Medicine
Stanford University
Stanford, Ca. 94305
(415) 497-6051

December 4, 1981

The Secretary
Federal Trade Commission
Pennsylvania Avenue and
6th Street NW
Washington, DC 20580

Dear Sir:

Re: File #792-3204. FTC staff report on the cigarette advertising investigation.

On behalf of the Stanford Heart Disease Prevention Program, I wish to applaud the scientific rigor of your May 1981 staff report on the cigarette advertising investigation, and to congratulate you on a thorough, well-researched and informative document.

As you have indicated, the American teenage and adult populations, especially smokers, are generally far from being sufficiently educated to make informed decisions about cigarette smoking -- quite apart from the addictive nature of cigarette smoking, which restrains even rational decisions.

This public ignorance accords with the Stanford Heart Disease Prevention Program's baseline findings in our two major California community education studies. It is evident that the cigarette manufacturers have failed to disclose many material facts about their product.

We strongly support your recommendations for rotating numerous specific warning messages, and favor a significantly enlarged arrow-and-circle format.

Sincerely yours,

JW Farquhar

John W. Farquhar, M.D.
Professor of Medicine
and
Director, Stanford Heart
Disease Prevention Program

JWF:sk

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

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Department of Epidemiology
and International Health

SAN FRANCISCO, CALIFORNIA 94143

December 1, 1981

Secretary
Federal Trade Commission
6th and Pennsylvania Avenues, N.W.
Washington, D.C. 20580

Re: File No. 792-3204

Dear Sirs:

I am a cancer epidemiologist, and one of my research projects involves an examination of trends in cigarette advertising directed at women over the past several decades in the United States, as well as a review of trends in female cigarette consumption patterns and disease rates.

I am writing in support of the summary recommendations of the Federal Trade Commission report of May 1981 entitled Staff Report on the Cigarette Advertising Investigation. The report's conclusions regarding the ineffectiveness of the current warning label on cigarette packages and advertisements is supported by recent Gallup and Roper polls showing that 13 to 17% of cigarette smokers in the United States are still unaware of the hazards of cigarette smoking to health. And yet these are the very people buying the cigarette packages where the "warning" label is printed. The current label is quite small relative to overall product or advertisement size, it is placed where it is visually least conspicuous, and the evidence suggests that it is too abstract to be understood by a sizeable segment of the population.

The public claim of cigarette manufacturers that advertising is not intended to convert nonsmokers to smokers but only to influence brand choice is belied by recent developments within the industry, including the increasing use of general circulation magazines and in particular women's magazines for cigarette advertising. By its own admission, the cigarette industry has targeted women as a vulnerable group for its existing and forthcoming ad campaigns. Advertising Age of September 28th in a front-page article titled "Women Top Cigarette Target" quotes Gerald H. Long, president/chief executive officer of R.J. Reynolds Tobacco as describing the women's market as "probably the largest opportunity for Reynolds." We know that reproductive risks (low birthweight and stillbirth) are increased in women who smoke and that lung cancer will become the number one cancer killer among women in the United States within the next few years, a trend due almost entirely to cigarette smoking. Yet, the number of pages of magazine advertising devoted to images of attractive, healthy-looking young women in cigarette promotions has increased manyfold over the past decade. For example, Better Homes and Gardens, the fourth ranking magazine in total circulation in the U.S. in 1980, now averages about 15 full pages of cigarette ads per monthly issue, and Ladies Home Journal (number nine in circulation) averages about 14 full pages per month.

It is difficult to explain to our children that while cigarette smoking is probably the number one health hazard in this country (accountng for a large proportion of cancer, coronary heart disease, respiratory ailments, deaths from fires, and other conditions), we allow cigarettes to be the most advertised product in the United States. If the advertising is to continue (a practice I do not condone), a true "fairness doctrine" is in order. I strongly concur with the recommendations of the Federal Trade Commission staff report that:

1. The current "invisible-because-so-familiar" warning label be replaced with rotational warnings of various sizes, shapes, and message content;
2. That the placement of the warnings on the ads be in a more noticeable location, where it can compete for the reader's attention;
3. That the written content of the warning be more concrete. (Instead of stating that cigarettes are "hazardous to health," it should state, "Cigarettes cause cancer," "Cigarettes shorten life expectancy," "Cigarettes increase your risk of dying of heart disease," "Cigarette smoking in pregnant women is related to low birthweight and stillbirths," and so on.)

Revenue comparable to that of the cigarette industry is not available to present a balanced view to the public, including the overwhelming scientific evidence of the health hazards of smoking. I urge that careful consideration be given to the well-conceived recommendations of the FTC staff report.

Thank you for your attention.

Sincerely,

Virginia L. Ernster

Virginia L. Ernster, Ph.D.
Assistant Professor of Epidemiology

VLE/as

cc: Peter R. Goldschmidt

TOBACCO SMOKE AND THE NONSMOKER: BIBLIOGRAPHY

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Jesse Steinfeld, M.D. — U.S. Surgeon-General, 1969 - 73
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Peter Pool, M.D. — President-Elect, American Heart Association, California Affiliate
Robert Fallat, M.D. — Director, American Lung Association of California
Charles Mittman, M.D. — Director, American Lung Association of California
Stanton Glantz, Ph.D. — Assistant Professor of Medicine, University of California, San Francisco
November, 1979

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Californians
for
SMOKING &
NO SMOKING
Sections

NORTHERN CALIFORNIA
HEADQUARTERS
P.O. Box 1061
Berkeley, CA 94701
(415) 841-2201

SOUTHERN CALIFORNIA
HEADQUARTERS
P.O. Box 45876
Los Angeles, CA 90045
(213) 671-1400

AMERICAN PUBLIC HEALTH ASSOCIATION
1015 Fifteenth Street, N.W., Washington, D.C. 20005 • (202) 789-5600

December 22, 1981

The Honorable Henry A. Waxman
Chairman
House Subcommittee on Health
 and the Environment
Committee on Energy & Commerce
2415 Rayburn House Office Bldg.
Washington, D. C. 20515

Dear Chairman ~~Waxman~~ Henry

President-Elect Tony Robbins, MD, has forwarded your correspondence concerning H.R. 4957, the "Comprehensive Smoking Prevention Education Act of 1981." The Association is pleased to support this important and needed legislation.

APHA recognizes the health hazards associated with smoking and has over the years participated in efforts to discourage and eliminate smoking. Internally, the Association accepts no tobacco advertising in its publications, holds no investments in firms which have a major interest in tocacco products, and allows no smoking at any of its meetings or public functions.

In our recent comments in response to the Federal Trade Commission's Staff Report on the Cigarette Advertising Investigation, we concurred with their findings that the public needs additional information about the health hazards of smoking. We agree that the current health warning is no longer effective. APHA supports your proposal to change the size and shape of warnings on cigarette advertising and packaging and to provide rotational warnings.

In addition, we endorse the proposal to provide statutory standing to the Office on Smoking and Health within the Department of Health and Human Services. APHA has recently expressed particular concern about funding cutbacks being experienced and proposed for the Office on Smoking and Health, and views support for this Office as one effective way to educate the public about smoking hazards.

We feel H.R. 4957 is an important step toward increasing public knowledge about the adverse health effects of smoking. We look forward to working with you and your staff in support of this legislation.

Very truly yours,

Stan

Stanley J. Matek, MS
President

MARYLAND STATE DENTAL ASSOCIATION
CONSTITUENT OF THE AMERICAN DENTAL ASSOCIATION

AIRPORT INVESTMENT BUILDING
793 ELKRIDGE LANDING ROAD
LINTHICUM, MARYLAND 21090

TELEPHONE: 796-4715

December 28,1981

Representative Henry A. Waxman
Chairman, Subcommittee on Health
and the Environment
Room 2415
Rayburn House Office Building
Washington, D.C. 20515

Dear Congressman Waxman:

This refers to your letter of November 25,1981, concerning H.R. 4957, the "Comprehensive Smoking Prevention Education Act of 1981".

As health care professionals, the members of the Maryland State Dental Association share your concern over the enormous toll in death and illness associated with cigarette smoking. We heartily endorse the proposed legislation. We feel that your efforts to prevent young people from smoking as well as to urge smokers to quit are positive steps to improve the health of the nation. We feel that public education is a necessary first step to public action to stop smoking.

Thank you for the opportunity to respond.

Sincerely yours,

Clayton S. McCarl, D.D.S.

Clayton S. McCarl, D.D.S.
President

CSM/pmt

Coalition for Health and the Environment

806 Fifteenth Street, N.W.
Suite 400
Washington, D.C. 20005

Telephone
(202) 737-5043

Honorary Chairman, Founder
The Honorable Paul G. Rogers

Executive Director
Arden C. Hyde

Executive Committee
President
Clyde E. Shorey, Jr.
Vice President for
Public Affairs
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Materials Program
Environmental Defense Fund

Michael F. Jacobson, Ph.D.
Executive Director
Center for Science in
the Public Interest

Stanley G. Karson
Director
Clearinghouse on Corporate
Social Responsibility

David L. Mallino, Ph.D.
Legislative Representative
United Steelworkers of America

Katherine S. McCarter
Assistant Executive Director
American Public Health
Association

Margaret Seminario
Industrial Hygienist
AFL-CIO

Myron G. Zeitz
Staff Attorney
Community Nutrition Institute

January 7, 1982

The Honorable Henry A. Waxman
House of Representatives
Washington, DC 20515

Dear Congressman Waxman,

The Coalition for Health and the Environment would like to express its support of your bill HR 4957, the "Comprehensive Smoking Prevention Education Act of 1981."

We strongly endorse the statutory protection of the Department of Health and Human Services' Office on Smoking and Health which your bill would provide. Our Coalition has been seriously distressed by the budget cuts the Office on Smoking and Health has received. We think the legislative branch needs to protect this, the only federal program addressing the major cause of the nation's most critical health problems.

The Coalition enthusiastically endorses your proposal that cigarette companies be required to use a variety of stronger, more specific health warnings on cigarette packs and in advertisements. We think that such an educational approach which promotes more informed consumer choices should be broadly embraced as a positive health promotion measure.

As the Federal Trade Commission has reported:

- Less than 3 percent of those exposed to cigarette advertising read the current printed warning, which has remained unchanged for over a decade.
- Although many people know of the link between smoking and lung cancer, more than 30 percent are unaware of the links between cigarette smoking and heart disease -- a far more prevalent killer of cigarette smokers.
- Nearly 50 percent of all women do not know about the risks of smoking during pregnancy.

We support your proposed legislation to strengthen the effect of cigarette health warnings by varying them and by making them more specific. As you know, smoking is a major cause of the nation's most prevalent and most serious diseases. We feel that HR 4957 is an important step to reducing the unnecessary health and economic costs smoking imposes on society. We look forward to supporting this legislation in any way we can.

Sincerely yours,

Ed Shorey

Clyde E. Shorey, Jr.
President

AMERICAN DENTAL ASSOCIATION
DR. ROBERT H. GRIFFITHS • PRESIDENT
1063 - 10TH STREET, BOX 177
CHARLESTON, ILLINOIS 61920

January 13, 1982

U.S. House of Representatives
Committee on Energy and Commerce
Subcommittee on Health and the Environment
Representative Henry A. Waxman, Chairman
Room 2415
Rayburn House Office Building
Washington, D.C. 20515

Dear Congressman Waxman:

I am pleased to read of your continued efforts aimed at the health risks associated with cigarette smoking. I share your concern and wholeheartedly support the intent of H.R. 4957. In fact, a growing body of evidence points to the relationship of smoking to the incidence and progression of periodontal disease which is fast becoming the biggest threat to optimum oral health in this country. We may want to add periodontal disease to the list of adverse health risks associated with smoking.

Unfortunately, from a dental health point of view, the health problems attributed to tobacco are not limited to smoking alone. We are most concerned with the rise in use of chewing tobacco, particularly by the young. While the risk of lung disease is greatly reduced, the risk of oral disease persists and may increase with the use of "smokeless tobacco." As you know, television advertising of smokeless tobacco products continues, often with celebrity endorsements. We need to look at this issue very carefully and very soon. Your support would be greatly appreciated.

Best wishes for a healthy and productive new year.

Sincerely,

Robert H. Griffiths, D.D.S.
President

RHG:lh

American Nurses' Association, Inc.
2420 Pershing Road, Kansas City, Missouri 64108
(816) 474-5720

Barbara L. Nichols, M.S., R.N.
President

Myrtle K. Aydelotte, Ph.D., R.N., F.A.A.N.
Executive Director

Cables
Amernurses U.S.A.

Washington Office
1030 15th Street, N.W.
Washington, D.C. 20005
(202) 296-8010

January 20, 1982

The Honorable Henry A. Waxman
Chairman, Subcommittee on Health &
  the Environment
House Energy & Commerce Committee
2415 Rayburn House Office Building
Washington, D. C. 20515

Dear Congressman Waxman:

In response to your recent request, we are pleased to comment on H.R. 4957 which you introduced to strengthen federal and private sector smoking education activities.

The American Nurses' Association applauds your action in sponsoring this legislation which constitutes a significant step on smoking prevention efforts. As the professional organization of the largest group of health care providers most of whom are women, we are especially concerned about the alarming increase in smoking among young women. The ANA encourages nurses to become informed about the health hazards of smoking and to be actively involved in health education programs particularly those to prevent young people from becoming smokers.

The American Nurses' Association supports H.R. 4957. We believe that Section 3 of H.R. 4957, which provides a statutory basis for the Office on Smoking and Health, is a necessary step especially in light of the Administration's attempt to cut funding in this area. The increased labeling requirements for cigarettes, as outlined in Section 4 of your bill, can only help to meet the need for increased public awareness of the devastating consequences of cigarette smoking. The enactment of H.R. 4957 will provide a much needed impetus to preventive health programs and stimulate new efforts to foster public awareness of the danger of smoking.

We look forward to continuing to work with you on your smoking prevention efforts. If we can be of further help to you, the staff of our Washington office will be happy to assist you.

Sincerely,

Barbara L. Nichols

Barbara L. Nichols
President

BLN:MM

American
Academy of
Pediatrics

P.O. Box 1034
1801 Hinman Avenue
Evanston, Illinois 60204
Phone (312) 869-4255
Chicago (312) 273-3846

Reply to:
Glenn Austin, M.D.
1080 Fremont Avenue
Los Altos, California
94022
(415) 948-6681

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February 5, 1982

Representative Henry A. Waxman
1721 Longworth House Office Building
Washington, D.C. 20515

Dear Representative Waxman:

On behalf of the American Academy of Pediatrics I am pleased to support HR 4957, the Comprehensive Smoking Prevention Education Act.

The human health consequences of cigarette smoking have been studied more throughly than those of any other environmental exposure. As was noted in the 1979 Surgeon General's Report, "Smoking and Health," specific mortality ratios are directly proportional to the years of cigarette smoking, and are higher for persons who started smoking at younger ages. As your legislation indicates, smoking contributes to mortality from lung cancer, cardiovascular disease and increases the risk of cancer from exposure to other carcinogens. Birth weight and fetal growth are also adversely affected by smoking during pregnancy.

Thus it is particularly alarming to pediatricians that despite our efforts to educate young patients about the dangers of smoking, the incidence of cigarette smoking is actually increasing among adolescent females, and has not decreased in young males.

The Academy applauds your initiative to combat this escalating problem and looks forward to assisting in whatever way you see fit.

Sincerely yours,

Glenn Austin

Glenn Austin, M.D.

GA:km

AMERICAN COLLEGE of PREVENTIVE MEDICINE
[illegible] FIFTEENTH STREET, NW SUITE [illegible] • WASHINGTON, DC [illegible] • (202) [illegible]

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Atlanta, Georgia
*EXECUTIVE COMMITTEE

February 25, 1982

The Honorable Henry A. Waxman
Room 2418
Rayburn House Office Building
Washington, D.C. 20515

Dear Congressman Waxman:

I am writing to express the endorsement of the American College of Preventive Medicine of H.R. 4957, a bill you have introduced which would help to provide information to the American people on the dangers of cigarette smoking.

By anyone's estimate, the annual cost of cigarette smoking to society and to individuals is enormous. Yet, a great many smokers are only vaguely aware of the consequences. The decision to smoke is one that an individual has a right to make -- in order to rationally make that decision, however, consumers must be provided additional information on the known hazards of the habit. Once given that information they will be better able to exercise their freedom of choice.

H.R. 4957 goes a long way in providing balanced information to those who may be unknowingly exposing themselves to tremendous hazards. We are, therefore, pleased to lend our support to this legislation.

Sincerely,

Jefferson C. Davis, M.D.
President

Association of
Teachers of
Preventive
Medicine

1015 - 15th Street, N.W., Suite 403
Washington, D.C. 20005

February 25, 1982

The Honorable Henry A. Waxman
Room 2418
Rayburn House Office Building
Washington, D.C. 20515

Dear Congressman Waxman:

I am writing on behalf of our organization to express our endorsement of H.R. 4957, a bill you have introduced which would change current labeling requirements for cigarettes and would make certain program changes within HHS to enhance the Department's anti-smoking efforts.

As the Surgeon General just this week reported, the dangers from smoking are far more extensive than we realized just a few short years ago. The concomitant health care costs associated with smoking are equally staggering. For these and other reasons, American consumers must be provided with far more facts than they currently are regarding the hazards of this habit. Your bill would make a substantial contribution in providing consumers with vital information they need in exercising their freedom of choice to smoke or not to smoke.

We are pleased to lend our support to this legislation aimed at ultimately curtailing the "chief preventable cause of death" in this country.

Sicnerely,

F. Douglas Scutchfield

F. Douglas Scutchfield, M.D.
President

AMERICAN CANCER SOCIETY, INC.
777 THIRD AVENUE • NEW YORK, N.Y. 10017 • (212) 371-2900

RESEARCH
EDUCATION
SERVICE

February 26, 1982

Honorable Henry A. Waxman
Chairman
Subcommittee on Health and the Environment
2415 Rayburn House Office Building
Washington, D.C. 20510

Dear Congressman Waxman:

The American Cancer Society, the world's largest voluntary health organization with over 2 million active volunteers in the United States, strongly endorses the basic purposes and provisions of H.R. 4957, The Comprehensive Smoking Prevention Education Act of 1981, and urges its early consideration and passage by the Health and Environment Subcommittee, the Energy and Commerce Committee, and the full House of Representatives.

Over 300,000 preventable deaths occur each year in this country because of cigarette smoking. Smoking is responsible for millions of hours of lost productivity costing our economy over $25 billion a year. It is a major cause of lung, larynx, oral cavity and bladder cancer.

A recent FTC study shows that despite efforts at education, the majority of our fellow citizens are basically unaware of the dangers of smoking. Education to the hazards of cigarette smoking, especially education efforts aimed at our young people who have not yet started to smoke are, therefore, vitally important. H.R. 4957 would take us a long way toward a coordinated, intensive effort at educating the American consumer to the actual dangers of smoking.

While some would question the efficacy of warning labels, the FTC staff report made it very clear that not enough information was getting to the public about the dangers of smoking. The sections of H.R. 4957 which provide for six rotating warning labels on all cigarette brands, labels which very specifically spell out the health hazards of smoking could well be the answer to this problem and must be tried.

The Society is particularly pleased that H.R. 4957 will now contain a provision requiring listing with the Secretary of Health and Human Services of all additives in each brand of cigarettes. Such a requirement could give scientists the opportunity to study the effects of burning and inhaling such additives on the health of cigarette smokers while still providing the tobacco industry with protection against revelation of trade secrets regarding quantities and types of these flavorings.

ACS is also in accord with the provision of H.R. 4957 which requires a formal Office of Smoking and Health as part of this package.

We wish to commend you for your leadership and foresight in offering H.R. 4957 and to thank you for your commitment to use education and research as a tool to reduce the national smoking habit thereby greatly reducing the number of preventable cancer deaths each year.

The American Cancer Society considers passage into public law of H.R. 4957 to be a major legislative priority. Therefore, if we can be of any assistance to you during the consideration of this legislation please do not hesitate to call upon us.

Sincerely,

Robert V.P. Hutter

Robert V. P. Hutter, M.D.
President

RVPH:rs

AMERICAN ACADEMY OF FAMILY PHYSICIANS

1740 WEST 92ND STREET • KANSAS CITY, MISSOURI 64114

OFFICE OF THE CHAIRMAN
BOARD OF DIRECTORS
DOUGLASS A. HADDOCK, M.D.
[illegible]
KALAMAZOO, MICHIGAN [illegible]

March 15, 1982

Honorable Henry A. Waxman, Chairman
Subcommittee on Health and
 the Environment
2415 Rayburn House Office Building
Washington, D.C. 20515

Dear Congressman Waxman:

The American Academy of Family Physicians would like to take this opportunity to voice its strong support for H.R. 4957, the Comprehensive Smoking Prevention Education Act of 1981. As an organization vitally concerned with the health and well-being of the people of this country, the Academy applauds your sponsorship of this legislation.

The Academy has long espoused the position that it is a desirable objective to keep healthy people well rather than simply provide their care once they have become sick. Indeed, continuous comprehensive care is one of the fundamental concepts on which the discipline of family medicine is based. However, each person must share in the responsibility for his or her own health. We believe H.R. 4957 will enhance the public's ability to assume responsibility for their personal health by increasing understanding of the substantial hazards of smoking. We hope and expect that this increased understanding will lead to a reduction in the incidence of smoking-related illness and death in this country, as well as reduction in the medical costs associated with such illness and death.

Again, we support your efforts and hope early passage of H.R. 4957 will be possible.

Sincerely

Douglass A. Haddock, MD

Douglass A. Haddock, M.D.

dah:ps

STATE OF CONNECTICUT
DEPARTMENT OF HEALTH SERVICES

OFFICE OF COMMISSIONER

March 17, 1982

The Honorable Henry Waxman, Chairman
House Commerce Health Subcommittee
The House of Representatives
Washington, D.C. 20515

Dear Congressman Waxman:

On behalf of the Association of State and Territorial Health Officials (ASTHO), I am writing in support of the provisions of H.R. 4957, the Comprehensive Smoking Prevention Education Bill. The issue of tobacco smoking and its health and social consequences must be addressed as a national problem. The results of tobacco useage in our country are tragic and in most cases preventable.

This bill is important and worthy of support for a number of reasons. The most important being that it is a comprehensive smoking prevention package. The bill has several major components:

1. It would establish an Office of Smoking and Health in the HHS Department This office currently exist but does not have legislative support. The office provides a valuable service to the nation and to individual states. Their service is unique and not available through any state or other federal agency. They provide technical information and assistance on a wide variety of topics dealing with the smoking issue.

   In the past, my staff has relied heavily on the resources of the office. The office's bibliographic service has assisted in answering questions from consumers, the medical community, and legislators. For example, the office helped track down information on the effects of chewing tobacco on the digestive system to assist a crewman on one of our Coast Guard stations. When we wanted to establish stop smoking classes through Connecticuts Adult Education Program, the office provided information on which classes were most successful in helping smokers quit. And, in 1979 when Connecticut was considering a Clean Indoor Air Law, the office supplied information on the provisions of other state's laws. No state health department or other federal agency has the staff or resources to duplicate this service.

2. It would establish an Interagency Committee on Smoking and Health. This provision makes good sense due to the complexity of the problem. It would enable more agencies and organizations to remain up to date on the issues and the current state of the art in prevention.

3. It would require the Surgeon General to transmit an annual report to Congress. Reports of this nature are extremely important and often are considered benchmarks of progress, as in the case of the 1964 report on Smoking and Health.

4. It would require cigarette companies to place health warning messages on each pack of cigarettes. I strongly support the rotating health message provision. The current health warnings on cigarette packages are not as effective as they could be. I believe that expanded educational efforts must take place and a system of rotating messages should be undertaken.

   A recent survey taken in the State of Connecticut indicated that 88 percent of a random samples of 500 state residents knew that smoking was harmful to health. Only 38 percent, however, recognized that smoking was a major risk factor for heart disease, which is the leading cause of death in Connecticut and the nation.

   Research has indicated that people will take preventive health actions when they perceive a problem as severe, consider themselves susceptible, and acknowledge a benefit from a remedial action recommended (Becker, 1974[1]). The strengthen labelling provisions address all of the necessary steps in the initiation of behavior change.

Finally, tobacco smoking is the number one public health problem in America. It is clearly the largest preventable cause of death in this country. This issue must be addressed as a national problem. H.R. 4957 addresses the topic at the national level and is a comprehensive well though out approach.

I urge the committee and Congress to support the bill.

Sincerely,

Doug Lloyd

Douglas S. Lloyd, M.D., M.P.H.
Commissioner

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[1] Becker, M., ed. The Health Belief Model and Personal Health Behavior. Health Education Monographs, (2) 1974, 324-473.

DSL:lz (3h)

Coalition on Smoking OR Health
A PUBLIC POLICY PROJECT WITH THE
NATIONAL INTERAGENCY COUNCIL ON SMOKING AND HEALTH
419 SEVENTH STREET, N.W., SUITE 401, WASHINGTON, D.C. 20004
FOR INFORMATION CONTACT
202/ 393-4446

[illegible] COMMITTEE
John D. [illegible], Chairman
American Heart Association
[illegible] C. Davis
American Cancer Society
[illegible]
American Lung Association
[illegible] Jr., M.D., Ph.D.
Chairman, National Interagency
Council on Smoking and Health

April 5, 1982

Honorable Henry A. Waxman
Chairman
Subcommittee on Health and the Environment
Committee on Energy and Commerce
Room 2415
Rayburn House Office Building
U.S. House of Representatives
Washington, D.C. 20515

Dear Congressman Waxman:

The Coalition on Smoking OR Health strongly endorses H.R.4957 and H.R. 5653 and requests that you include in the record of the hearings of the Subcommittee on Health and the Environment on these bills the following materials which add further to the mountain of evidence already before the Subcommittee which demonstrates both the need for and merit of this legislation.

1. Cohen and Srull, "Information Processing Issues Involved in the Communication and Retrieval of Cigarette Warning Information", November 1980;

2. Keenan and McLaughlin, Inc., Cigarette Warning Project, May 1981;

3. Whelan, et al., "Analysis of Coverage of Tobacco Hazards in Women's Magazines", Journal of Public Health Policy, March 1981;

4. Smith, "The Magazines' Smoking Habit", Columbia Journalism Review, 1978.

Sincerely,

Matthew Myers
Matthew L. Myers
Staff Director

INFORMATION PROCESSING ISSUES INVOLVED IN THE COMMUNICATION AND RETRIEVAL OF CIGARETTE WARNING INFORMATION

Joel B. Cohen
Thomas K. Srull

Center for Consumer Research
University of Florida

November 1980

I. Introduction

An appropriate public policy goal for cigarette warning information would seem to be that this information should be made available for evaluation by consumers at or around the time a purchase decision is made. Accordingly, one alternative would be to provide appropriate information at the time and place of purchase, which consumers could then consider in making a purchase decision. However, the complexities of cigarette distribution (e.g., the number and diversity of sales outlets and mechanisms) suggest that there may be difficulties associated with choosing any one particular remedy. For example, the existing warning information on packaging may not be sufficiently prominent to engage the consumer's attention during a shopping trip and, in fact, may not even be accessible until after the purchase decision is made. Nevertheless, since this important warning information is critical in making a rational consumer decision, alternative means of providing such information near the time a purchase decision is made should be reviewed.

Evaluating potential advertising vehicles in light of this policy goal requires an understanding of the factors that combine to affect subsequent retrieval of this information when a purchase decision is being made. By way of comparison, it should be understood that advertising is only one part of the overall marketing program for a product. Considerable effort goes into point of purchase marketing activities (e.g., product packaging, in-store advertising, premiums and discounts, personal selling), much of which is explicitly designed to remind the consumer of information stressed in the advertising. One function of in-store tie-ins is to aid in the retrieval of information from past advertising so that the key "reasons to purchase" are made salient at the optimal time. Thus, from the perspective of the total marketing program, influencing memory storage and retrieval through a combination of advertising and in-store promotion is a well-practiced art.

It is also important to realize that the in-store "information environment" is characterized by competing brand messages, which present positive reasons to select one brand over another. This type of environment almost exclusively provides cues to stimulate and aid retrieval of brand-based attributes and images. Warning messages from advertising, which are not directly linked to particular brands, are less likely to be spontaneously recalled by consumers when in-store cues lead them to retrieve brand-based associations. Put another way, if a piece of information (such as a warning) is not specifically stored with a particular brand category, it is not likely to be retrieved by a fairly casual and hurried search of information in that category. Thus, a general warning message placed in an advertisement will have great difficulty competing for information retrieval with brand-based information at the point of purchase.

To sum up, even a well presented product warning message is at an obvious disadvantage relative to "pro-brand messages", as the former relies much more heavily on the spontaneous recall of information from memory than the latter. Weaker presentations, of course, place a heavy burden on the often harried shopper to spontaneously examine their stored knowledge. This disadvantage is accentuated when the product is one that is purchased almost habitually rather than contemplatively. Therefore, in addition to examining possible point of purchase information disclosure mechanisms, the Commission may also need to consider methods of strengthening the initial warning disclosure appearing in cigarette advertising in order for it to have any chance at all of competing with skillfully presented and possibly powerful appeals developed for each cigarette brand. That is true not only for initial attention in an advertisement but also for retrieval at the point of purchase.

II. Criteria Appropriate for Evaluating Warning Effectiveness

Determining appropriate criteria for evaluating whether warning information conveyed in advertisements is in fact recalled at the time of a purchase decision is obviously a critical issue. One of the most important things to note is that survey questionnaires are one of the least effective, and potentially one of the most misleading, techniques for determining whether warning information is actually accessible for use in making a purchase decision. There are several reasons for this. First, survey questionnaires really assess prompted or "cued" recall in which people respond to specific questions or cues. This situation is not at all identical to one in which a person is able to spontaneously recall information about a particular brand or product class. Advertisers implicitly recognize this when they provide in-store cues that are intended to help people retrieve previously acquired information.

In general, memory researchers typically make a fundamental distinction between "availability" and "accessibility" (see Tulving & Pearlstone, 1966). Once information is fully comprehended and encoded into long-term memory, it is thought to always be "available" (Lewis, 1979). That is, there are psychological mechanisms in the brain that permanently store and retain such information in the absence of some profound effect on specified regions of the brain. However, only a small portion of the vast quantities of information that we learn is "accessible" at any given time. That is, we are only capable of retrieving a fraction of the total information we have available. More importantly, it is widely recognized that information that is accessible in one context will not be accessible in another. Similarly, information that is accessible at one time will not necessarily be accessible at another time (either earlier or later).

Probably the two most important determinants of whether information is accessible at any given time are: (1) the amount of competing information in the same "content domain" that we have also learned, and (2) self-generated and externally-generated retrieval cues that are present at that particular time. A simple example of how these processes operate may be helpful. Most people "know" the name of their first grade teacher and have that information "available." That is, once the information is learned it is probably never "forgotten" in the sense of not being preserved in the brain cells of the central nervous system. However, each of us learns the names of literally thousands of people by the time we are adults. Thus we may not be able to retrieve or spontaneously recall the name of our first grade teacher in later years due to the large number of competing responses. External retrieval cues will help. If we are shown old photographs or told the name of other old teachers or classmates, we will be more likely to retrieve the name. Internal retrieval cues will also help. If we pause to reminisce about our old school and continue to think about our teacher and classmates, we will also be more likely to retrieve the name. Special techniques like hypnosis that allow us to form rich images of past events and people will also help us to retrieve the name. If we are successful in retrieving the name, however, it is likely that we will once again "forget" it or be unable to recall it at some later time. In general, any message that an adult fully comprehends and encodes into long-term memory will be "available" from that point on. However, such information will only be "accessible" in a limited set of circumstances.

There are two well documented phenomena in the experimental psychology literature that are relevant to these issues. First, spontaneous or "free" recall is not conceptually equivalent to probe or "cued" recall--there is a great deal of information that people are able to retrieve when provided with specific cues that they are unable to retrieve in the absence of any cues. This means that responses to survey questions indicating that people remember that a warning message is present in cigarette advertisements can not be taken as evidence that this information is accessible at the time of purchase (i.e., in an environment in which relevant cues are not specifically presented).' The second point is somewhat related. Specifically, people do not typically make judgments or decisions that are based on all of the relevant information that is stored in memory. Rather, only a small subset of the information is typically used.

The fact that people can retrieve information in response to specific cues that they are unable to retrieve in the absence of such cues is exemplified in a classic study by Tulving and Psotka (1971). These investigators examined a memory phenomenon that is relevant to advertising called "retroactive interference." Retroactive interference refers to the fact that later learning typically inhibits the recall of previously acquired information. Tulving and Psotka reasoned that learning later material could interfere with previously acquired information through at least two mechanisms. First, subsequent learning could result in a general decay or weakening of the memory traces associated with earlier items. Alternatively, subsequent learning may have no effect on earlier traces but it may make it more difficult for a person to independently access or retrieve previously learned information (perhaps by making it more difficult for the person to independently produce relevant cues). To test these hypotheses, Tulving and Psotka presented subjects with various numbers of word lists. Each list contained 24 words, consisting of four words from each of six semantic categories (e.g., types of buildings). The category names themselves were not provided. Some subjects learned the target list and then recalled as many words as possible. Other subjects learned the target list and then learned from one to five other (interpolated) lists before attempting to recall words from the original target list. The results of the study were quite clearcut. First, the number of words recalled from the target list consistently declined as the number of interpolated lists increased. These results are consistent with those of may other studies showing the effects of retroactive interference. However, it still would not be clear whether such results were due to a weakening of the original traces or to some retrieval failure. Tulving and Psotka addressed this question by administering a second (cued) recall test in which category cues for the six semantic categories were also presented. When the cued recall procedure was used, the number of words recalled from the target list was virtually unaffected by the number of interpolated lists. These results indicate that traces for the original items were still "available" and the "forgetting" associated with the number of interpolated lists was really due to an inability to retrieve the original items. They also indicate that information that can be retrieved in response to externally-provided cues can often not be retrieved when such cues are not provided. Thus, information that is available is not necessarily accessible. It is also important to note that these general findings have been replicated many different times in many different contexts (see e.g., Buschke, 1973; Tulving & Pearlstone, 1966). They have also led many contemporary theorists (e.g., Eysenck, 1977) to propose that virtually all "forgetting" is due to retrieval failure. Information that continues to be available simply becomes less accessible without the aid of relevant retrieval cues.

It is important to realize that the crucial role of retrieval failure is not confined only to performance on memory tasks. Tversky and Kahneman (1973, 1974; Kahneman and Tversky, 1972, 1973) have demonstrated in a series of papers that retrieval processes also play an important role in human judgment and decision-making. Without going into detail, these authors have demonstrated that people do not perform an exhaustive search of memory for all relevant information in order to make a particular judgment or decision. Rather, people typically base their judgments on only a subset of this information that happens to be most accessible at the time. Thus, those factors that are most easily retrieved are most likely to be used in making a particular decision. To the extent that advertising campaigns result in unique brand associations being most accessible, and to the extent that in-store cues make brand-based information most likely to be retrieved, more general product warning information is not likely to be salient when making a purchase decision.

All of this research is consistent in leading to several general conclusions concerning the criteria that should be used in evaluating the effectiveness of (existing or proposed) warning information. Whether consumers are likely to consider such warning information in making a decision at the point of purchase is a question of accessibility. Survey questionnaires or any other assessment devices that contain specific probes simply examine whether such information is potentially available. They do not assess whether warning information is able to be independently retrieved and they are inappropriate for drawing conclusions concerning the accessibility of the warning message. More appropriate criteria would involve less structured tasks. For example, consumers at the point of purchase might simply be asked to report any factors that came to mind when considering a particular decision. Note, particular information may come to mind and yet be largely ignored as a basis for the decision. We see demonstrated accessibility of warning information as an appropriate criterion for the FTC, but not persuasion (i.e., changing consumers' attitudes toward smoking or evaluation of "health hazards") under present regulatory auspices. Such data, then, would provide a more accurate "baseline" of the extent to which warning information is actually accessible when making a purchase decision. An alternative method might be to ask people to recall all of the information related to cigarette advertising as possible. Although this technique also involves a direct probe, it is a relatively general and weak one. The advantage of this method is that the researcher could not only examine the percentage of people who mention the warning message but, by examining the order in which information is recalled and the total amount of time needed to provide such information, a general measure of the saliency of such information is also provided. One would expect that: (1) warning information might only be spontaneously recalled by a small percentage of consumers, and (2) even for these people, it is not likely to be terribly salient at the point of purchase (i.e., one would expect such information to come out late in the protocol and take a relatively long time to generate).

In summary, retrieval may be thought of as the end point of a process that begins with attention and encoding of a stimulus. Therefore, factors that enhance attention to information and allow for elaboration and the formation of cognitive associations will increase the likelihood of unprompted recall.

III. Factors that Enhance Elaborations During Encoding

Retrieval factors are obviously very important to the study of consumer information processing, as well as to the specific issues of concern in this paper. However, to enhance the likelihood of retrieval, policy makers need to concern themselves with the overall process that begins with the allocation of attention to various aspects of the stimulus field and which proceeds through the formation of more or less elaborate stimulus associations during encoding. Tulving and Thompson (1973) have discussed in detail the extent to which encoding and retrieval factors are conceptually interdependent.

One recent model of encoding and retrieval processes, that is relevant to several issues of concern in this paper is the levels of processing framework introduced by Craik and Lockhart (1972). Perhaps the single most important reason that this is important is that it emphasizes that information can be encoded in many different ways, and these will have strong effects on the ability of subjects to subsequently recall such information. In other words, the same stimulus can be processed in many different ways, and subsequent memory for the same information will largely be a function of the way in which it was encoded in the first place. In considering possible remedies, it will therefore be important to consider not only certain stimulus factors (e.g., presentation format) but also the types of encoding strategies that are likely to be evoked by different types of information.

The levels of processing framework has been enormously heuristic in cognitive psychology and much of the research generated by it has been discussed in Cermak and Craik (1979). We will describe only one of the many types of experiments that have been conducted to demonstrate how the same stimulus can be processed in extremely different ways. Craik and Lockhart initially identified several simple experimental tasks that could be used to induce different types of processing. For example, a subject might be shown a series of adjectives, some of which are presented in small letters and some of which are presented in capital letters. The subject's task is simply to decide whether the word is printed in small letters or capital letters. Note that subjects need not even silently pronounce the word to perform such a "structural" task. In other conditions, subjects might be shown a particular word and have to decide if it rhymes with another target word. Such a "phonemic" task requires that a subject pronounce the word but it does not require that any of the semantic implications of the word be extracted. Craik and Lockhart also used a "semantic" task in which subjects would be presented with a particular word and have to decide whether it was a synonym of a separate target word. Such a semantic task requires that the subject extract the full semantic implications of the word. Craik and Lockhart hypothesized that these tasks make increasingly intense processing demands on the part of the subject and an identical word will be processed to different "levels" depending upon the processing strategy of the subject. Moreover, they demonstrated that words processed to deeper levels would subsequently show far different levels of recall. That is, given an identical stimulus list of words, subjects performing semantic processing recalled significantly more than those performing phonemic processing, and these in turn recalled significantly more than those performing structural processing. It should be emphasized that these differences (and others that have been subsequently examined) are extremely robust and generally quite large. Tulving (1979), for example, concludes that "in terms of the sheer magnitude of the effects, encoding operations must be regarded as among the most important determinants of memory performance" (p. 410).

It was once thought that semantic tasks evoked the most extensive processing and produced the highest levels of recall. Rogers, Kuiper, and Kirker (1977), however, have identified an even more powerful type of encoding strategy. These authors used a "self-reference" task in which subjects had to decide whether a particular word described themselves. They also used all of the original Craik and Lockhart tasks as comparison conditions. Rogers et. al. found that the structural task produced the least extensive processing and lowest levels of recall, followed by the phonemic task and then the semantic task. Thus, they replicated the Craik and Lockhart findings. Rogers et. al. also found, however, that the self-reference task produced by far the most extensive processing and the highest levels of recall.

It is, therefore, clear that exactly the same stimulus can be processed in a variety of ways, and the encoding strategy one uses to process a particular piece of information will largely determine how well it is subsequently recalled. More important, specifically relating a piece of information to oneself seems to induce deeper levels of processing than any other known task or type of strategy.

These general results have also been replicated many times. Their implications are further explored when we discuss the effects of making information personally relevant (see below). Their similarity to one other interesting finding in the attitude change literature should be noted however. Specifically, persuasive communications seem to have a similar property in that it does not seem to be the stimulus message per se that is crucial but the cognitions on the part of the subject that are generated by a particular communication. Greenwald (1968), for example, discusses evidence indicating that the cognitions generated while listening to a persuasive communication are a better predictor of subsequent attitude change than the number of arguments presented in the communications per se. Again, getting a person to think about the personal implications of information seems to be an extremely effective strategy.

In sum, it is clear that stimulus factors will be important in considering possible remedies to the problems of concern in this paper. It is equally clear, however, that the same stimulus message can be processed in a variety of ways. In particular, large memory differences can be produced by inducing subjects to adopt different encoding strategies. Specifically, determining the personal implications of information for oneself appears to induce extremely extensive processing, and this should be an important consideration in choosing among various possible remedies. Many of these issues are discussed further in the following section.

IV. Factors that Influence the Retrieval of Information and the Use of Information in Making a Purchase Decision

Fortunately, there is a rich empirical literature bearing on those factors that increase or decrease the likelihood that information will be retrieved in a given context. The sheer quantity of research in this area makes a thorough review of the literature beyond the scope of this paper. In fact, several book length reviews have recently appeared (e.g., Bransford, 1979; Brown, 1976; Eysenck, 1977). Rather than attempting to be exhaustive, this section will concentrate on those factors that are most relevant to advertising and the communication of existing warning information.

Novelty. One of the most potent factors in increasing the likelihood that a piece of information will be spontaneously retrieved is novelty. Information that is novel or unexpected seems to capture one's attention, is processed more extensively, and subsequently is much more likely to be recalled than information that is redundant or expected to appear in a given context. For example, von Restorff (1933) found that almost any technique that served to increase the novelty of particular items or led them to be unexpected enhanced the subsequent recall of those items This has since become known in the memory literature as the "von Restorff effect" and Hastie (in press), Srull (1980), and Wallace (1965) have recently reviewed literally hundreds of studies that have consistently replicated this same basic effect. The fact that enhanced recall for novel information has been found with bigrams (Smith, 1973), nonsense syllables (von Restorff, 1933), words (Jenkins & Postman, 1948), complex action sequences expressed in written prose (Bower, Black, & Turner, 1979), courtroom trial evidence (Reyes, Thompson, & Bower, 1980), written descriptions of personal behavior (Srull, 1980), and filmed sequences of interpersonal behavior (Hastie, 1980) indicates that it is an extremely robust retrieval phenomenon.

In this regard, it is worth noting that existing warning information is redundant in both form and content. That is, the warning labels contained in cigarette advertisements and placed on cigarette packages have not changed in nearly a decade and have never contained any new information. Moreover, years of redundancy have presumably led smokers and nonsmokers alike to expect such labels on all cigarette-related materials. Interestingly, memory researchers have also known for some time that novel information not only captures more attention and is better recalled than redundant information, but it does so at the expense of other (redundant) information in the display. For example, Newman and Saltz (1958) found that novel items not only showed enhanced recall but the immediately surrounding items in the list showed unusually low levels of recall. These results suggest that one's attention is drawn to novel information. However, since one's attention and processing capacity is limited, this necessarily means that less attention can be paid to immediately surrounding information. Since advertisements are continually changing and often contain novel verbal and pictorial material, it is not surprising to find that many people report not even seeing the warning label when looking at standard advertisements.

One final point needs to be made concerning these expectancy or novelty effects. Srull (1980) has recently demonstrated that although unexpected information shows clearly superior levels of recall, there is virtually no difference between one's ability to recognize expected and unexpected information that was previously presented. These results are important because the major difference between a recall task and a recognition task is that only the former requires a subject to retrieve an item in the absence of any cues. Thus, it is thought that any variable that has an effect on recall but not on recognition taps a process that is localized in the retrieval stage of information processing (see e.g., Anderson & Bower, 1972, 1974; Bahrick, 1969, 1970; Eysenck, 1977; Glass, Holyoak, & Santa, 1979; Kintsch, 1968, 1970; Watkins & Gardner, 1979). It is also important to note that the recall-recognition differences found by Srull were obtained with the same subjects. Thus, this is a clear indication that people are able to produce redundant information in response to external cues that they are unable to retrieve in the absence of such cues (see also Graesser, Woll, Kowalski, & Smith, 1980).

In sum, cigarette advertisements are continually changing and often contain novel verbal and visual information that is likely to capture one's attention. In contrast, existing warning information has not been changed in years, does not contain any novel information, and is not likely to elicit much attention. Although such warning information may be recognized or recalled in response to direct probes, this is irrelevant since it is not likely to be spontaneously recalled at the point of purchase. To the extent that the point of purchase environment stimulates one to retrieve brand-based attributes and/or make a hurried decision (see above), the likelihood of spontaneously recalling warning information is even further reduced.

It should be noted that several relatively simple techniques could be used to keep warning labels relatively distinctive and novel. These techniques are concerned with novelty both within advertisements and between advertisements. For example, the print style could be varied so that it is different from any text material presented in the actual advertisement and also different from (many) other warning messages (i.e., print style could be varied across specific ads as well). Varying the nature and color of the surrounding boarder could also be easily accomplished (von Restorff [1933] found that both print style and a change in color could be used to make items distinct). In addition to these stylistic changes, however, changing the semantic content of the warning message (i.e., have a variety of semantically different warning messages) is probably the single most powerful means of keeping this information novel and distinct.

Concrete and abstract information. There are a number of issues related to the way in which people differentially process concrete and abstract information. Concrete information generally refers to single objects or events that are readily transformed into mental images. In contrast, abstract information generally refers to abstract concepts that are not readily transformed into mental images. For example, a picture is obviously concrete since it already contains a specific visual image. The issue is more general however. For example, the words "automobile" and "cigarette" are also very concrete, as it is very easy to form mental images of such objects. On the other hand, words such as "justice" or "hazardous" or "health" are very abstract and quite difficult to transform into mental images. The distinction between concrete and abstract information can also be applied to more complex types of information. For example, a picture of a cancerous lung would be very concrete, while a statistical summary of the number of deaths each year due to cancer would be very abstract.

The evidence is now overwhelming that people form mental images and that such imagery has a number of important consequences (for a very recent review, see Kosslyn, 1980). First, concrete information is better remembered than abstract information. This general conclusion has been supported in a number of different ways. For example, pictures appear to be much easier to remember than words (Mandler & Johnson, 1976; Mandler & Parker, 1976; Paivio, 1971, 1978a, 1978b; Shepard, 1967). Also, concrete or high-imagery words are more easily remembered than abstract or low-imagery words (Paivio, 1971). Another potentially important effect was discovered in the literature on paired-associate learning. Specifically, concrete words serve as more effective cues than abstract words (i.e., a concrete word will be a better retrieval cue for information that is associated with it than will an abstract word).

These findings have a number of interesting implications for the present area of concern. For example, many cigarette advertisements contain rich photographic information. One would hypothesize that such pictures are processed to a far greater extent and are better remembered than the accompanying text. Similarly, concrete information involving specific people and events would be expected to be better recalled than abstract information that does not contain any specific referent. In this regard, it is important to consider that existing warning messages are *extremely* abstract in nature. Not only do they contain abstract words that are hard to visualize like "hazardous," "health," and "concluded," but they do not contain even one specific piece of concrete evidence on which the abstract conclusion is based. Finally, even if people once knew specific research findings concerning the dangers of cigarette smoking, such abstract warning labels would not be very effective cues for eliciting such information. In contrast, the concrete pictures and labels that are often used in advertising, packaging, and in-store displays would be expected to be much better cues in eliciting previously learned information about the associated brand.

Concrete and abstract information are very important in human judgment and decision-making as well as in memory. A rather large literature has accumulated in recent years indicating that people rely on concrete information to a much larger degree than they rely on abstract information in making judgments or decisions (see e.g., Nisbett & Ross, 1980; Nisbett, Borgida, Crandall, & Reed, 1976; Ross, 1977). In some cases, these differences are dramatic. Nisbett and Borgida (1975; Borgida & Nisbett, 1977) report an extremely powerful tendency for subjects to manifest an overreliance on concrete and a corresponding underreliance on abstract information quite unlike the "rational economic man" generally assumed to be the decision-maker. For example, subjects virtually ignore abstract descriptive information about a population of people in predicting the behavior of a single individual. On the other hand, subjects very readily use the behavior of a single individual to predict characteristics of the entire population (see also Hamill, Wilson, & Nisbett, 1980). Interestingly, Slovic, Fischoff, and Lichtenstein (1976) have found that similar processes operate when people estimate the risk associated with various activities or events; abstract statistical summaries are largely ignored, while vivid individual cases are weighted quite heavily. Kates (1962) also notes how people seem to ignore the abstract information contained in actuarial tables and he has discussed the "inability of individuals to conceptualize floods that have never occurred" (p. 88). Clearly, information processing limitations and seemingly unrecognized (i.e., by people themselves) biases in the use of particular types of information lay to rest the notion of "unbounded rationality" in decision-making. These traditional assumptions can no longer be used as a basis for policy making without doing violence to the prevailing evidence. It might be very useful, therefore, to develop some direct evidence as to how people estimate the risk of health hazards such as lung cancer from various types of warning labels and how such information is subsequently processed.

It is also interesting to note that advertisers often use pictures of unusually vigorous and healthy-looking individuals who ostensibly live full and rewarding lives untroubled by any "hazardous" effects of smoking. This sort of a portrayal of a smoker is of course somewhat inconsistent with any personal manifestation of deleterious effects of smoking. Thus the warning information may appear inconsistent with the "actual" effects of smoking on individuals with whom the consumer might identify. The research evidence presented above suggests that readers are easily prone to draw conclusions about the general population of cigarette smokers from these sorts of concrete examples. Existing research would certainly suggest that they are more likely to be used than the abstract warning labels.

Motivational factors and the personal implications of information. The personal implications of information received and motivational factors impinging upon the person who receives such information are two factors that are each somewhat related to those discussed above. Furthermore, they will often have powerful interactive effects. Without going into the technical details of specific experiments here (several of which were discussed earlier), a number of recent studies have demonstrated the effects of making information personally relevant to a subject. Markus (1977), for example, has demonstrated that personally relevant information is attended to more readily and processed more easily than personally irrelevant information. A series of studies by Rogers and his colleagues (e.g., Kuiper & Rogers, 1979; Rogers, Kuiper, & Kirker, 1977) has also shown that information is better recalled when it is personally relevant. There is also very strong evidence indivicating that concrete information is perceived to be more personally relevant than abstract statistical information (Borgida & Nisbett, 1977).

In general, it appears that abstract information is more likely to be perceived as personally irrelevant than concrete information. Moreover, this is complicated by the role of motivational factors. In particular, there is a great deal of evidence to suggest that people are likely to dismiss as irrelevant evidence that is highly discrepant with their own behavior or beliefs. For example, persuasive communications containing information highly discrepant with one's prior behavior or attitudes are often perceived as less accurate, less informed, and more illogical (see e.g., Dietrich, 1946; Hovland, Harvey, & Sherif, 1957; McKillop, 1952). As one might expect, they typically have very little effect on the person's own beliefs or subsequent behavior (see e.g., Fisher & Lubin, 1958; Hovland et. al., 1957; Insko, Murashima, & Saiyadain, 1966; Whittaker, 1964a, 1964b). In addition, this tendency to ignore relevant information that is discrepant with one's own beliefs and/or behavior is strongest with high involvement issues such as one's own health (Freedman, 1964). In this regard, it is also interesting to note that beliefs in the accuracy of the initial Surgeon General's report were lowest among those people who smoked most heavily (Kassarjian & Cohen, 1965). It must be acknowledged, therefore, that for many smokers (especially long-term and heavy smokers) any warning message is apt to be somewhat threatening. This is particularly true if the person believes he or she is not able to stop smoking. Under these conditions, many smokers will probably deny, distort, or otherwise ignore the warning information and possibly block it from recall. It may be quite difficult to design a message that would be readily retrieved by such people, but it is clear that to have any chance of being effective it must be responsive to the information processing issues raised here.

In sum, information that is perceived to be most personally relevant will be best remembered. Although there is no direct evidence for this, there is every reason to believe that this will be true for both brand-related information and warning information. In general, however, concrete information will have a larger impact on one's decisions than abstract information. This would appear to put existing warning information, relative to brand-specific information, at a severe disadvantage in terms of the likelihood that it will be spontaneously recalled. This is complicated by the fact that people who have already smoked (or, perhaps, even those who are already predisposed toward smoking) are relatively unlikely to perceive the abstract warning message as personally relevant (and relatively likely to perceive specific brand-related information contained in cigarette advertisements as more personally relevant). Finally, it should be emphasized that the motivational factors discussed above can be extremely powerful. In all honesty, it is probably safe to assume that they can at best be minimized but not eliminated by virtually any type of change in message tactics. Nevertheless, an explicit attempt to make the warning information more personally relevant would at least help.

All of these factors make an overall recommendation concerning possible remedies very delicate. On the one hand, making warning information more personally relevant should result in better memory for such information on the part of consumers and also should result in such information being taken into account when making a purchase decision. On the other hand, information concerning dangerous health hazards is more likely to raise levels of anxiety and therefore be defended against and dismissed from consideration precisely when its personal relevance is emphasized. This role of direct personal relevance on the processing of information has long been examined in work on persuasive communications (for a summary, see McGuire, 1969). Although there is a definite motivational tendency for one to reject arguments that are highly discrepant with one's own behavior and prior attitudes, two factors that, in combination are capable of counteracting this have been identified. First, the message is much less likely to be ignored or dismissed when the source (speaker) is a high-prestige, knowledgeable "expert" in the area in question. This should pose no particular problems in the present area of concern. The second important factor is that some positive step that can be taken to "solve the problem" also needs to be included. In other words, a message that arouses fear or anxiety also needs to include positive steps that can be taken to reduce that anxiety (see e.g., Hovland, Janis, & Kelly, 1953; Leventhal & Niles, 1964). In the present context, making information concerning the dangers of cigarette smoking more personally relevant should be accompanied by information that it is indeed possible for one to stop smoking. In addition, further information concerning the health benefits of stopping would also be helpful. Much of the research relevant to these points is summarized in Leventhal's (1965, "Fear communications in the acceptance of preventive health practices.") Finally, it might also be noted that although single communications are capable of producing attitudinal changes, they probably are not sufficient to produce actual behavioral changes. For example, Leventhal, Watts, and Pagano (1967) used high fear arousing communications and low fear arousing communications, and measured both intentions to quit smoking and actual reductions in the number of cigarettes smoked. High fear arousing communications were more effective in terms of intentions to quit smoking, but there was no significant difference in terms of actual reduction in smoking between groups exposed to the two types of communications. We can only speculate as to what impact there might have been on actual smoking behavior with a longer running program having the full endorsement of the leading medical and health authorities in the country.

Retrieval cues. Another extremely important class of determinants of how likely it is that information will be recalled in a given context are the retrieval cues present in the situation. The availability-accessibility distinction and the important research of Tulving and Psotka (1971) discussed above are both obviously relevant. There is, however, one other very important factor that needs to be considered.

As discussed above, relevant retrieval cues can be used to enhance recall for information that is not accessible in the absence of such cues. The key word, however, is "relevant." Not all retrieval cues are equally effective and, in fact, some cues will be completely ineffective. Consider, for example, a prototypical verbal learning experiment in which subjects are presented with items that are drawn from several different semantic categories. Providing the category names will obviously enhance recall. That is, people will be able to recall more of the information when presented with the category names as retrieval cues than when they are not presented with such cues. However, providing a single category name as a cue will only enhance recall for items within that category; it will not increase the probability of recalling items from other categories, in spite of the fact that they were presented in the same list (Tulving & Pearlstone, 1966). Since packages and displays are unique to a particular brand, their presence at the point of purchase is likely to cue and enhance the recall only of information that is specific to the associated brand. However, recall of information that is not brand-specific, such as that obtained from a warning label, is not likely to be enhanced as a result of those types of retrieval cues being present in the purchase environment. In fact, to the extent that a habitual or hurried decision is made and/or a hurried memory search is performed, recall of information that is not directly associated with the retrieval cues present may even be suppressed.

Such findings, of course, are only a special case of Tulving's (1979; Tulving & Thompson, 1973) "encoding specificity" principle. This principle is concerned with the more general finding that only those features of the information that are considered at the time of encoding will be effective as retrieval cues at some later time. Similarly, any time a person subjectively organizes information into several discrete categories, a cue related to a single category will only aid the retrieval of information that is specific to that category.

The implications of this for the present issues of concern are clear. First, people typically obtain cigarette-related information from a series of discrete advertisements. This information is acquired at different points in time and each advertisement generally contains information about only a single brand. It is therefore likely that people organize cigarette-related information according to brand-based attributes. It is also likely that warning information is generally not considered to be brand-specific. To the extent this is true, warning information will not be organized with brand-based attributes. As noted above, in-store tie-ins generally provide retrieval cues at the point of purchase. However, such cues are related to specific brands and will enhance the recall of brand-based attributes, and probably will not be effective in making the warning information more accessible. Thus, on the basis of current experimental data, one would hypothesize that warning information is relatively unlikely to be recalled for use in making a purchase decision. Also as noted above, the more brand-specific cues there are in the environment and the more hurried a consumer's decision, the less likely it is that warning information will become accessible at the point of purchase.

Interestingly, organizational strategies have been shown to be extremely important in determining how likely it is that information will be recalled but they are relatively unimportant in how well such information is recognized (Kintsch, 1970; McCormick, 1972). This indicates that the effects of such organization are localized in the retrieval stage of information processing. Once again, if people organize information according to specific brands, this will have a pronounced effect on the type of information they are able to recall but little effect on the type of information they are able to produce in response to specific probes.

V. Potential Remedies and Criteria Appropriate to their Evaluation

Our discussion thus far has a number of direct implications for the evaluation and development of warning messages. The purpose of this section is to summarize a few of the key points and to suggest several ways in which the factors we have identified can potentially be applied. Several specific points have been made about the criteria to be used in evaluating remedy effectiveness. First, warning information must be able to compete with information about specific brands at the point of purchase. Second, there must be some evidence that warning information is "accessible" for use in making a purchase decision. Third, obtaining responses to survey questionnaires or other assessment devices that provide direct probes is not appropriate to assessing how likely it is that warning information will be spontaneously recalled or considered at the point of purchase. Research from both the memory and judgment literature supports this.

It was argued above that what is needed is a more indirect technique. Asking people at the point of purchase to report those factors that came to mind when considering a purchase decision is one alternative. Another alternative is simply to ask people to recall (retrieve) as much information as possible from cigarette advertisements. One could then examine the order in which information is recalled and the time required to make a particular response to determine how salient warning information is to the typical consumers. In addition, it would be valuable under any proposed presentation strategy to assess whether the warning information is differentially salient to smokers and nonsmokers and to different categories of smokers (e.g., young vs. older, heavy vs. light). This might provide valuable information for consideration of a possible mix of approaches.

The general criteria outlined above are equally appropriate for evaluating the effectiveness of existing warning information and any potential changes that might be instituted. It should also be noted, however, that experimental analogues can be used to determine a priori which of several possible remedies is likely to be most effective. Since any significant changes in remedies may involve some length of time commitment to a particular course of action, the use of prior empirical research would seem to be prudent.

The research cited earlier suggests several ways in which existing warning information could be changed to make it more effective. Although we will briefly outline several suggestions, it should be emphasized that their actual effectiveness is really an empirical question. It is our belief that sound empirical research can be used to guide the Commission in selecting from a wide array of possible remedies those that are most likely to be helpful to consumers when making their decisions.

One of the major problems with existing warning information is that it states a very general, abstract conclusion and provides no specific information on which that conclusion is based. Footnote 283 on page 84 of the recent Staff Report indicates that an extremely small percentage of the general population has any access at all to this type of information. One possibility, therefore, would be for warnings to contain more specific,concrete and personally relevant information. The research cited above suggests that such information is more likely to attract attention, be processed further, and be more easily retrieved.

A related issue that also was discussed above concerns novelty. In short, the warning information has not been materially changed in nearly a decade while cigarette advertisements remain distinctive by continually changing. The experimental data discussed above suggest that warnings would be more effective if they also contained new information and were presented in a variety of formats. In general, a variety of specific warnings that changed across various advertisements should capture more attention and ultimately have more of an effect in terms of being accessible at the time of purchase. In this regard, it should be noted that basically the same informational content can be presented in a number of different ways (indeed, advertising firms are confronted with just such a problem). For example, a warning that vividly shows the reduced life expectancy of a person who smokes one pack of cigarettes a day for 10 years and a separate warning that gives the reduced life expectancy for another individual who smokes a half a pack a day for 20 years are providing partially redundant types of information. Nonetheless they are more novel, distinctive, likely to be perceived as more personally relevant, and more likely to be used in maing a purchase decision than a single abstract conclusion presented on two different occasions. As advertisers are well aware, frequency of exposure is not nearly as important as the way in which people think about the information presented (cf. Greenwald, 1968).

Parenthetically, it may be worth investigating whether some of these warnings should simply contain an address from where a more detailed report of the possible health hazards associated with smoking could be obtained. This would have the added benefit that subsequent short warnings would be likely to cue the more elaborate information contained in the larger report. In general, a warning that will activate more specific information that is already stored in memory will be most effective.

One final issue also needs to be considered. There are going to be important differences, at both the cognitive and motivational level, between people who already smoke and people who do not. While warning information contained in cigarette advertisements may well be the best way of getting smokers to consider such information in making their purchase decisions, we believe that separate campaigns (not tied to cigarette advertisements) concerned with the dangers of smoking will be especially more effective with those who do not already smoke. The latter individuals may have very different reasons for smoking, some of which can be confronted quite directly (and probably effectively) by approaches targeted specifically to the needs and concerns of this consumer segment. A combination of these two approaches would therefore probably be most appropriate and effective for the population in general.

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Prepared By:

Keenan & McLaughlin Inc.
New York, N.Y.
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CIGARETTE WARNING PROJECT

I. GENERAL INTRODUCTION

Keenan & McLaughlin Inc., a full-service, New York City advertising agency, has been retained by the FTC staff to develop a warning plan for use in advertising by cigarette manufacturers. Specifically, we have been asked to examine the feasibility and costs, from an industry perspective, of a program that would require the use of a new warning symbol or symbols, and an as yet unspecified number of new health warnings, to be placed within the symbol(s).

A) Description Of Keenan & McLaughlin Inc.

Keenan & McLaughlin Inc., founded in 1970, is a member of the American Association of Advertising Agencies. Keenan & McLaughlin Inc. creates and places advertising in the consumer, agricultural and industrial product areas for clients such as: ABU-GARCIA (fishing gear); Bass Ale (beers and ales); Burberry's (coats); Callard & Bowser (candies); Dow Chemical U.S.A. (agricultural products; MEM (toiletries); Mobil Oil Corporation (consumer and industrial promotions); and, Orrefors, Inc. (imported crystal).

The people who have worked on this report are Mr. Michael E. Keenan, Chairman of the Board, Mr. Frank J. Byrne, Jr., Vice President/Management Supervisor, and Mr. Paul Richey, Vice President/Media Director. Mr. Keenan has 21 years of advertising agency management experience at Fuller & Smith & Ross, Foote Cone & Belding, and Compton Advertising. Mr. Byrne has 30 years of experience, having worked for Farm Journal, CIBA-GEIGY, Lewis & Gilman, Fuller & Smith & Ross, Popular Science, and the Morristown (New Jersey) Daily Record. Mr. Richey joined Keenan & McLaughlin Inc. from McCaffrey & McCall, Inc., where he was a Vice President and Media Director. Prior to that, he was with the National Outdoor Advertising Bureau for 10 years.

The sources for this report include: Advertising Age (advertising billing figures); Business Week; Leading National Advertisers, Inc.; Marketing and Media Decisions; The Bureau of Advertising of the Newspaper Publishing Association; The Magazine Publishers Association; Standard Rate & Data; Institute of Outdoor Advertising; and the Association of National Advertisers. In addition, the agency has had experience in the production and placement of millions of dollars of advertising materials for magazines, newspapers, billboards, transit, point-of-purchase, and promotion materials during the past decade.

B) Summary Of Recommended Rotational Warning System

We have developed our recommendation to examine the feasibility and costs of requiring the cigarette companies to disclose in each advertisement one of an as yet undetermined number of warnings.

The staff of the FTC gave us several guidelines:

First, the plan had to provide for the maximum dissemination of all the warnings in all advertising, thereby ensuring that the public would be exposed to all the warnings.

Second, we were to develop a plan which would be the least costly and least burdensome to the cigarette companies.

Third, we were to develop a plan which did not discriminate in favor of or against any media which carried cigarette advertising. We were told by the staff that it did not want to discourage the use of cigarette advertising or the use of any particular medium by cigarette advertisers.

Fourth, the plan was to be structured so as not to interfere with the introduction and withdrawal of brands.

Fifth, to the extent possible, the plan had to fit in with the companies' existing way of advertising.

Sixth, the plan had to be one which would allow the companies to demonstrate compliance with the plan without incurring unduly high costs.

After careful consideration of numerous alternatives, we have concluded that the most efficient way to accomplish these objectives would be to create a single symbol within which separate health warnings would be inserted. The FTC could provide the artwork for the warnings and symbol to be used in the advertising. In the alternative, the cigarette companies could create their own artwork as long as it meets or exceeds specifications issued by the Commission.

Under this plan, each cigarette manufacturer would rotate the health warnings in all advertising for their brands. The manufacturers would assign each brand a specific order of warnings to include in its advertising. The advertising for each brand would display the appropriate warning for a three-month period and then switch to the warning next in order on the list to which that brand was assigned. Every three months, all advertising for the brand would carry a new warning. Once all warnings on the assigned list had been displayed for the three-month period, the advertising would return to the warning at the top of the list, and continue through the warnings on that list, changing the warning each quarter.

There are two exceptions to this plan. For billboards, the companies would be asked to devote a small percentage of their entire outdoor advertising budget to billboards which would carry just the warning symbol and the messages assigned to each of their brands during that three-month period. In return, no warnings would be required in their billboard advertising for the products themselves.

For point-of-purchase and other promotional material, the month in which the material is ordered will determine the message to be carried.

II. BACKGROUND: THE PREPARATION AND PLACEMENT OF CIGARETTE ADVERTISING

The preparation and placement of cigarette advertising essentially involves four types of participants: cigarette companies; advertising agencies hired by these companies to create and place advertisements; the media in which the advertisements appear; and, other miscellaneous suppliers, such as firms setting type, making engravings, taking photographs, retouching photographs, producing point-of-purchase materials, and printers.

A) Cigarette Advertisers

Each of the cigarette companies has a Product Manager for each brand or group of brands. The Product Manager is responsible for coordinating the advertising effort of that brand. While the Product Manager has various staff support people (e.g. media, marketing, research, etc.) available at the company, he normally relies on the advertising agency for the creation and placement of advertisements for that brand.

Based on the company's research, a marketing plan for each brand is created. The Product Manager then prepares a communications plan with specific objectives and strategies for his specific brand(s). The Product

Manager works closely with his primary advertising agency contact person in the development of this communications plan. Many, but not all, of the creative needs of the cigarette company (based on this communications plan) are turned over to the respective advertising agencies for each brand for execution.

B) Advertising Agencies

The cigarette companies retain a number of advertising agencies to create and place cigarette ads in the media. The agencies each work on one or more of the company's brands. The services, described in detail below, are executed separately for each brand or group of brands. Within agencies, the various services include:

1. Initiation. The Product Manager at the cigarette advertiser informs the Account Manager at his agency that specific advertisements are needed to fulfill certain objectives of the communications plan.
2. Creation. The Account Manager interprets these requirements to the agency's assigned Writer/Art Director team and Media Planner by a work requisition.

The Writer/Art Director team discuss the objectives and create an advertisement, in rough form, for the Account Manager to present to the client. The Media Planner prepares a media plan based on demographics of the audience sought to respond to the advertising message.

3. Approvals. The Account Manager presents to and secures from the advertiser's Product Manager approval for both the advertisement and the media plan.
4. Production. The Account Manager informs the Writer/Art Director team of this approval. They, in turn, arrange for completion of the elements of the finished advertising. The Production Manager supervises the production of the advertisement and purchases the actual printing materials which will be sent to the publications.
5. Traffic. A Traffic Manager works with the Account Management people and the Media Planner to establish the exact "placement" of the advertisements within each medium. For instance, he establishes a "Schedule Flow Chart" indicating where each print advertisement will appear in magazines and newspapers. Separate schedule flow charts are prepared for both

transit and outdoor markets, specifying which advertisements are to appear in each market at a specific "showing" (circulation) level over a specific time period. With the agency Production Manager, he sees that the correct printing materials are sent to the correct media with explicit instructions regarding when they should appear.

6. Implementation. Based on the approval of the client, the Media Planner purchases the advertising space in the media. A "Schedule Flow Chart" showing where the advertisement is to appear (in relationship to other advertisements scheduled for the brand) is supplied to the Product Manager at the cigarette advertiser, so he can determine that the advertisement will appear in each media at the right time, as budgeted.

C) Media

The major media used by cigarette manufacturers include:

1. newspapers and newspaper supplements;
2. magazines;
3. billboards and outdoor signs;
4. transit and sidewalk posters;
5. point-of-purchase materials; and
6. promotional materials.

As explained above, the selection of media is determined on a brand-by-brand basis, depending on the communications need of each particular brand or group of brands. There are several steps taken in the preparation and placement of the advertising material that is sent to the various media for reproduction and printing of each advertisement. These steps, by medium listed above, are as follows:

1. Newspapers and Newspaper Supplements

   The advertising agency has a "mechanical" assembled from the various components of the advertisement including type, illustration and warning notice, and sends it to an engraver who makes offset film. This offset film is then converted to a Velox (an actual photographic print of the finished advertisement), which, in turn, is sent to each individual newspaper where it is used as the basis for the actual printing materials for black and white advertisements.

   The agency media department sends contracts (space commitments) and insertion orders (specific instructions for an individual advertisement) to the newspapers in which the cigarette manufacturer has authorized the purchase and placement of cigarette advertising. The agency accounting department will receive invoices and proof of performance (tearsheets of each ad published) from the publication, pay the media, and bill the cigarette manufacturer. The cigarette manufacturer advertising department checks the agency invoices against the schedule authorized and instructs their accounting department to pay the agency.

2. Magazines

Three methods of printing are used by magazines (including theatre programs) - offset; letterpress; and, rotogravure:

a) Offset. The "mechanical" and original photography or artwork is sent to an engraver who makes offset film. This film is sent to the magazine and is used as final printing materials to make the press plates.

b) Letterpress. The engraver makes letterpress (copper) plates from the "mechanical" and original photography or artwork and these are sent to the magazines for use as final printing material for the press plates.

c) Rotogravure. The agency sends the "mechanical" and original photography or artwork to a rotogravure house where roto separations are made. These separations and color proofs are then sent to the publication and are used to make the final printing materials, press plates. *Sunday supplements (e.g. Parade, New York Times Magazine) commonly use the rotogravure process.

The agency media department sends contracts (space commitments) and insertion orders (specific instructions for an individual advertisement) to the magazines in which the cigarette manufacturer has authorized the purchase and placement of cigarette advertising. The agency accounting department will receive invoices from the magazines and proof of performance (tearsheets of each ad published) from the magazines, pay them, and bill the cigarette manufacturer. The cigarette manufacturer advertising department checks the agency invoices against the approved schedule and instructs their accounting department to pay the agency.

3. Billboards and Outdoor Signs

There are essentially two kinds of outdoor signs - poster panels and painted bulletins. The panel is 12'3" high by 24'6" long and holds three different size standardized printed posters. The painted bulletin is usually 14' by 48' and is custom designed.

a) Poster Panels. "Mechanicals" are sent to specialty printers where the billboards are printed in sections (sheets). These printed (and numbered) sheets are then sent to the outdoor company (who owns or leases the actual billboards and sign locations) for posting (pasting up) on boards in each "outdoor" market. They are sold on a monthly basis and the paper is also changed monthly.

b) Painted Bulletins. Advertisements are painted directly on the boards by the outdoor company (who owns or leases the actual billboards and sign locations) using the agency supplied materials as a guide. The boards are usually contracted for on an annual basis, they are painted once and the advertiser usually receives three repaintings a year.

Outdoor advertising is sold on a market-by-market basis. The outdoor company, or "plant," owns or leases the sites of the outdoor advertising. Within each market, the advertiser buys panels and bulletins from the plant in terms of population exposure to the message. For example, poster panels are sold monthly by gross rating points (G.R.P.) or "showings." A 100 G.R.P., or showing, is the number of panels needed to expose the message to 100% of the population of the particular market each day. A 50 G.R.P. or showing delivers a daily exposure to the advertiser's message equivalent to one-half of the market population. Bulletins are usually sold by the year rather than the month, and are usually sold on the basis of an individual location.

The agency media department sends contracts (space commitments) and insertion orders (specific instructions for a "showing") to the outdoor posting companies in each market that has been authorized by the cigarette manufacturer to be purchased. The agency accounting department will receive invoices and proof of performance (posting statements) from the posting companies and bill the cigarette manufacturer. The cigarette manufacturer advertising department checks the agency invoice and posting company statements against the list of previously authorized locations and then instructs their accounting department to pay the agency.

4. Transit and "Sidewalk" Posters. This medium is similar to outdoor in that the advertising space is owned or leased by a plant, and that the space is sold by G.R.P. and "showings." The unit of purchase here,

just as in outdoor, is the month, and the ad itself is normally re-posted monthly.* The advertising agency sends a "mechanical" to a printer who prints cards and ships them to the transit advertising company. The transit advertising company physically "posts" the cards in accordance with the media plan, within trains and train stations, inside and outside of busses, and in bus shelters.

The agency media department sends contracts (space commitments) and insertion orders (specific instructions for a "showing") to the transit posting companies in the markets that have been authorized by the cigarette manufacturer to be purchased. The agency accounting department will receive invoices and proof of performance (posting statements) from the transit posting companies, check them against the specific showings ordered, pay the transit posting companies and bill the cigarette manufacturer. The cigarette manufacturer advertising department checks the agency invoice and transit posting company statements against the list of previously authorized showings and then instructs their accounting department to pay the agency.

5. Point-of-Purchase Materials. This is advertising displayed around the areas where cigarettes are usually sold and generally consists of signs posters, rubber counter change mats and other such items. The advertising agency supplies "mechanicals" and/or individual art elements to the specialty firms who design and produce point-of-purchase materials. These items are ordered in certain quantities as they are needed and supplied to the cigarette advertiser's sales force for distribution and placement. They are in place at retail outlets for extended periods of time, often for years in the case of some items. When additional quantities are needed, another order is made, sometimes entailing a revised message on the item.
6. Promotion Materials. Duplicates of the same art elements used in the cigarette brand's advertising are supplied to specialty manufacturers who produce various promotion materials such as match books, cigarette lighters, beach towels, plastic and non-plastic "tote" bags, calendars, T-shirts, etc., that are used to promote that particular brand of cigarettes. These items are sometimes purchased by the consumer at a price less than its usual

retail value when accompanied by proof-of-purchase (wrappers, carton ends) of that particular brand. Like point-of-purchase materials, these are ordered from specialty manufacturers in specific quantities and are produced at one point in time. As more items are needed, they are ordered again, often with a revised advertising message.

D) Art Suppliers

Also involved in the production and placement of cigarette advertising are other miscellaneous supplier organizations who perform various services, usually directly for the advertising agency. Some of these firms are:

Supplier Firm	Function
Commercial Photographers	Take photographs.
Commercial Artists	Create artwork for use as illustrations.
Photographic Retouchers	Retouch photographs before final use.
Type House	Supply various styles and sizes of type.
Art & Mechanical Studios	Assemble artwork, photography and type in a "mechanical" format for engravers and rotogravure houses to make final production materials. This function is sometimes performed by advertising agencies themselves.

Supplier Firm	Function
Engravers	Prepare - 1) engravings from above for use in letterpress publications, and, 2) offset material, i.e. film, from above for use in offset publications.
Rotogravure Houses	Prepare rotogravure production material from above for use in publications using this method of printing.
Lithographers	Prepare printed material for use in outdoor.
Printers	Printing brochures, point-of-purchase materials and material for transit posters.

III. PROPOSED ROTATIONAL WARNING SYSTEM

The program that we recommend is designed to cause the fewest administrative problems and the least cost to the cigarette manufacturers, their advertising agencies, the media carrying the advertising, the various suppliers involved in the production of advertising, and to the Commission in its compliance monitoring program.

As will be explained in greater detail below in our discussion of the costs of a rotational warning plan, significant costs can be saved by allowing sufficient lead time for the advertisers to introduce the rotational warnings in new advertising. We recommend, therefore, that the date on which a rotational warning plan becomes mandatory be delayed. In addition, advertisers should be given the option of including their first rotational warning in any new advertising introduced between the date the rotational plan is announced and the date it becomes effective. In this way the warnings can be incorporated in the ad in the pre-production stage, a far less costly alternative than placing a new warning in an existing advertisement.

In addition to minimizing costs, the program is designed to insure the warning system will reach a maximum number of consumers in an effective way. As indicated above, we are assuming that the Commission will provide the text of approved warnings. Within the guidelines provided us by the staff (pp. 2-3, supra), we have considered a number of rotational plans. Some of the issues we examined include:

- Should all brands from a particular company carry the same warning or should each brand from the same company carry different warnings?

We recommend that the rotation occur on a brand-by-brand basis, for the reason explained in part A.

- Should the warnings change each month, each quarter, each year, with each new ad execution, or with each new ad campaign?

For reasons explained in Part B, we recommend that the warnings on each brand change quarterly.

- Should all cigarette advertising be treated the same or should it be treated differently, according to the media in which it appears, or form in which it appears?

For the reasons stated in Part C, we recommend that all advertising except for that on billboards and promotion ad materials contain rotating warnings. For billboards, we recommend that a percentage of the brand's outdoor advertising expenditures be devoted to billboards carrying just the specific warning for that brand during that quarter. For promotional materials, we recommend that the warning appearing on such material be determined by the date on which the material is ordered.

- Should there be one warning symbol or more than one symbol?

For reasons stated in Part D, we recommend that all warnings be rotated within a single symbol.

- Should the warnings that are target-specific be placed in media aimed at that target?

While the placement of warnings that are target-specific in media particularly aimed at that target might be the ideal, we recommend that the warnings rotate in all media equally for the reasons stated in Part E.

A) Rotation By Brand

We considered several different ways to rotate warnings. The options considered were:

- having all advertising carrying the same warning at the same time.
- having warnings rotated cigarette manufacturer by cigarette manufacturer, so that advertising of all brands of a single manufacturer would carry the same warning.
- having warnings rotated brand by brand.

We rejected the first option because public exposure to all of the warnings would be delayed for a considerable length of time. Also, each warning under such a system would be seen only very infrequently.

We recommend rotation of warnings by brand rather than by cigarette manufacturer for the following reasons:

First, by having different warnings assigned to different brands, all of the warnings will be in the marketplace at all times. If we had chosen to rotate warnings on a "company" basis, only six warnings could appear at any one time. Under a quarterly rotation, and depending upon the number of warnings, rotation by company would mean that some warnings would not appear for a significant length of time.

Second, rotation by brand will insure that each warning will receive approximately the same level of exposure at all times. Rotating by company would mean that the warnings assigned to the smaller cigarette manufacturers, or those who advertise less, would receive less exposure over the short term.

Third, rotation by brand lessens the likelihood that a particular warning would be associated with any one company.

Fourth, rotation by brand adds few additional administrative or financial burdens to the cigarette companies. Advertising is usually developed for each brand separately and each cigarette company ordinarily maintains separate marketing and administrative staff for each brand, or closely related groups of brands. (Example: Menthol "lights" and Menthol "ultra lights".)

Rotating by brand would operate in the following way.

Six different schedules are to be prepared, one for each company. Each of the six companies will select a schedule at random. Each schedule contains as many lists as there are warnings, with each list containing all the warnings. For instance, assuming there are 16 warnings, each schedule includes 16 lists and each of those 16 lists would have a different order of the 16 warnings.

Schedule "A" would begin with its first list beginning with warning number 1. Schedule "B" would begin with its first list beginning with warning number 4; Schedule "C" with warning number 7; Schedule "D" with warning number 10; Schedule "E" with warning number 13; and Schedule "F" with warning number 16.

The company selecting Schedule "A" would assign the first list, which begins with warning #1, to its first brand. It would assign the second list, which begins with warning #2, to the next brand, and assign the third list, which begins with warning #3, to the third brand.

If the company had more than 16 brands, it would start over and assign the seventeenth brand with the list beginning with warning #1.

The company that receives Schedule B would assign the list beginning with warning #4 to its first brand, the next brand the list beginning with warning #5, its third brand the list beginning with warning #6 and so forth.

The company using Schedule C would assign the list that begins with the seventh warning to its first brand, its next brand the list beginning with warning #8, and its third brand the list beginning with warning #9.

SCHEDULE A

Brands	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Warning	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1
	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2
	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3
	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4
	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5
	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6
	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7
	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8
	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9
	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10
	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11
	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12
	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13
	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15

SCHEDULE B

Brands	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Warning	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3
	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4
	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5
	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6
	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7
	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8
	10	11	12	13	14	15	16	11	2	3	4	5	6	7	8	9
	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10
	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11
	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12
	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13
	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1
	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2

When a cigarette manufacturer introduces a new brand, it would assign it the next list in succession. New brands, in test market situations, would also be assigned lists in this same manner.

B) Frequency of Rotation

In order to provide for the maximum disclosure of the warnings with the least cost and burden to the cigarette companies, we had to balance exposure of the warning versus added costs to the cigarette manufacturer. Options studied included:

- Rotation monthly.
- Rotation quarterly.
- Rotation annually.
- Rotation with the introduction of new advertising.

Monthly rotation would impose greater administration, production and monitoring costs on both the cigarette manufacturer and the FTC. It would, however, reduce the chance that one brand would be associated with any one warning.

On the other hand, a proposal for changing warnings on an annual basis has the disadvantage of running the possibility of associating one brand with one warning. In addition, the public would receive less exposure to to those warnings assigned to brands that do little or no advertising.

A third option was to change the warning with each new advertising execution or advertising campaign. There are several problems with this option. First, it would be very difficult to monitor. Ad campaigns are of varying duration and it is often difficult to determine when a specific campaign begins or ends. Unless cigarette manufacturers were required to inform the Commission when each new ad campaign was to begin and end, the Commission would not be able to monitor compliance without a substantial expenditure of resources. Second, given the varying duration of ad campaigns, some warnings would run for only a brief time while others would run for very long periods. Thus, in some cases some warnings would receive little exposure, while others might become overly identified with a particular brand. Third, by controlling the duration of their ad campaigns, cigarette manufacturers would control how much exposure each warning would receive. This would leave too much discretion with the cigarette manufacturers.

With a quarterly rotation system, the warnings would rotate frequently enough for wide dissemination without risk that any one warning would be overly associated with one brand. A quarterly warning rotation system would also be less costly to administer and monitor than a monthly warning system, since there will be only four warning changes per brand per year.

C. Rotation System By Media

Having decided to recommend a system of quarterly rotation of health warnings by brand, we considered how best to implement this system in each medium in which cigarette advertising appears. We will explain in detail below our recommendations for each medium, including a discussion of alternatives considered and rejected. The basic system had to be modified for outdoor advertising, and for promotional and point-of-purchase material, due to special problems of rotating warnings in those media.

1. All Print Media: Newspapers, Newspaper Supplements, Magazines, And Theatre Programs

The same warning that is assigned to a particular brand from its warning list, would appear for a three month period in all print advertising for that brand. We recommend that the "cover date" of each publication determine which warning is to appear. Warnings in ads in inserts would be determined by the "cover date" of the publication in which the insert appears.

An alternate option was to determine the appropriate warning message based on the date(s) on which the publication actually "appears" on newsstands or is delivered to subscribers. The date on which various publications actually "appear" varies from publication to publication. Some publications actually appear in subscriber's homes and on newsstands a few days and sometimes several weeks (e.g., monthly magazines) prior to the date shown on their cover. All publications, of course, can be in circulation after their "cover date." Therefore, the simplest proposal is one using "cover date" as the controlling factor determining exposure of the warning message. Using "cover date" is compatible to the way print advertising generally is purchased and prepared.

2. Transit And Sidewalk Posters

The quarterly warning message assigned to each brand would also appear in transit posters and sidewalk posters (bus shelter, etc.) displayed during each monthly posting period for that brand. Transit and sidewalk posters are usually purchased on a monthly basis; new messages or new paper with the same message are normally posted once each month. Therefore, no additional cost is incurred when changing warning messages on an ongoing basis. Each quarter, when the posting is changed anyway, the replacement posters would carry the next assigned warning.

3. Point-Of-Purchase And Promotional Materials: Mail Circulars, Give-Away Items

We recommend that the warning assigned by the cigarette companies to these items be the scheduled warning in effect for each particular brand during the month in which the items are ordered. Once the order for these items has been placed, the warning on each item need not be rotated if the item is displayed or given away in a different quarter. If a point-of-purchase or promotional item is re-ordered, then the date of reordering would govern which warning would appear. This system will make it easier to monitor and administer. It will also minimize the costs to the cigarette manufacturers because items of this nature are generally produced all at one time, even if their use will last more than one quarter.

Alternate options considered were to have these media carry only one, standard warning message or to carry the message(s) scheduled for the quarter(s) while the item was in use. The use of one message was rejected since this militates against an even distribution of all warnings among the population over time. Since these items are traditionally produced/printed in a single production run, yet have a distribution life of varying and often indeterminate length, it would be more difficult and more expensive for both the advertisers and the FTC to require the message to vary quarterly.

Another alternative considered was to have no warning message required at all. This option was rejected because it was felt that it could tend to artificially create a shift of advertising expenditure emphasis within this industry towards these media.

An additional alternative considered was for each production run of a promotional or point-of-purchase item to include an equal number of items carrying each warning. Assuming, as above, that there would be 16 warnings, each 1/16 of every production run would carry a different warning. This was rejected for the obvious cost penalty this sort of program would impose upon the cigarette manufacturer.

A manufacturer, under this system, could "select" the message to appear on these items by pre-selecting the date of ordering. While this is obviously true, it is doubtful that, in fact, manufacturers will find one warning in general more conducive to cigarette sales than any other. In addition, in the long run, as the cigarette manufacturers continue to produce these items, all messages will be used.

4. Billboards And Painted Displays

Because of problems related to inadequate exposure of a warning in this medium, we considered a number of options to create a system that would best provide for meaningful exposure of the warnings yet without undue cost to the cigarette manufacturers. We recommend that a percentage of each brand's monthly billboard and painted display advertising expenditures be allocated to the production and placement of billboards containing only the new symbol and scheduled warning message, with the rest of the brands' billboards not carrying any warning message or symbol. We feel that this system would most effectively meet the guidelines established by the FTC staff.

Our reasons are these:

The distance from which one views billboards is between 100 to 400 feet. The speed at which one is moving when the message is seen also limits the number of words that can appear on a billboard and still be read and understood. The current rectangular warning, for example, is virtually invisible to the occupants of a vehicle moving 55 mph, 200 to 300 feet away. It is important, in creating outdoor advertising to keep the message short enough that it can be understood in the short time that a viewer sees it, and large enough that it can be seen.

We considered many alternatives. One was to omit warnings entirely from all outdoor advertising. This was rejected because of the current large volume of cigarette outdoor advertising. More billboards advertise cigarettes than any other product. In addition, it would probably result in a switch of advertising dollars from the other media to outdoor.

Another alternative was to enlarge a warning to make it visible and understandable. However, we feel that increasing the size of a warning to the point where it is readily visible may result in the warning taking up a substantial amount of the billboard's space. This might deter the cigarette manufacturers from advertising in this medium and would interfere with the way in which they ordinarily advertise. The danger of interfering is particularly likely if any new warnings are as long as or even longer than the current warning. We do not, of course, know the length of any new rotational warnings. If they were short enough, it might be possible to include them in outdoor advertising without unduly interfering with the advertiser's message. Additional research would be useful to clarify this possibility.

A final option, and one we recommend, is to have the warnings placed on separate billboards. This would ensure that the messages are effectively exposed, without undue cost to the advertisers. Each cigarette company would be requested to spend 5% of its monthly outdoor budget for the production and placement of warning message billboards. The messages produced would depend upon each of that company's individual brands' rotational schedules.

The showing allotment for the warning message billboards would have to be in proportion to each plant's distribution of non-illuminated and illuminated signs and would have to be equal to or better than the audited exposure values for panels of each type within each plant.

We had originally thought that the best way to operate a system of separate billboards would be to require an allocation based on showings or G.R.P's. Requiring merely a percentage of a brand's billboards to carry the warning message could have resulted in an unequitable distribution of warning message billboards and, unlike a system based on showings, would have limited the advertiser's flexibility. An allocation of warning billboards based on showings would have been difficult to monitor, and neither that system, nor one based on a percentage of billboards, could account for the differences between printed posters and painted bulletins.

Using a percentage of expenditure system permits the equitable handling of both painted displays and outdoor posters without giving any preference to advertisers who use more of one type or the other. It would also lessen the likelihood of a shift within the outdoor industry itself from one type of outdoor sign to another.

The system based on a percentage of outdoor expenditures would be much easier to administer than one based on showings yet it would not sacrifice reach and frequency for the warning messages. This expenditure allocation system assures proper carriage for the warning messages without leaving loopholes which could result in an unequitable warning message distribution.

The key phrase is that the warning showing allotment must be in proportion to each plant's distribution of non-illuminated and illuminated signs and must be equal or better than the audited exposure values for panels of each type within each plant.

These audited exposure values for each outdoor plant are available in Traffic Audit Bureau (T.A.B.) reports or from the plant operators themselves, and the plant invoices for each month's postings can be readily checked against this document during compliance monitoring.

D. Number of Symbols

We considered whether there should be rotating symbols as well as rotating warning messages. While rotating symbols might increase the novelty of the system and, at least at first, generate greater consumer attention, the added costs appear to outweigh any potential benefits. The costs to the cigarette companies would rise because each different design could require separate sets of warnings that would have to be produced and administered, complicating production schedules and increasing the costs of monitoring the warning system itself. If there is one symbol, changing the warnings is a simple operation. The ad can be left intact with only the language inside the symbol being changed. When the symbol itself must be changed the costs are several times greater. (See costs discussion, infra).

Moreover, there is a value in having cigarette health warnings associated with a single symbol: Over time, a distinctive symbol may have a health warning benefit in itself.

E. Specific Warnings In Specific Media

One of the possibilities we considered was to have specific warnings appear in highly selective media. For example, a higher percentage of warnings dealing with smoking and pregnancy would appear on women's publications. Another example would be birth control warnings in publications aimed at young women. Although this might add to the effectiveness of those particular warnings, it also might result in a shift of cigarette advertising dollars from these publications. Therefore, we recommend against placing target-specific messages more heavily in the media directed at that target audience.

IV. COSTS TO CIGARETTE MANUFACTURERS TO IMPLEMENT PROGRAM

The costs of the rotational system that we have recommended are at most minimal. In this report we will provide background information on current advertising costs for representative units of each medium discussed in this report, with the exception of point-of-purchase and promotional materials for which this information is not available. We will then provide estimates on the production and administrative costs to the cigarette manufacturer of including a new symbol and warning in advertising that has already been produced without them; changing the warning after the new symbol is included in the advertising; and incorporating the new symbol and warning in "new" advertising.

We have also included a section on the costs to the FTC for the creation of a new symbol, setting the type for 16 different health warning messages to be used within the symbol, and producing reprints to be distributed to the cigarette companies to be used as artwork in their advertising. The cigarette manufacturers could also be given the option of using FTC symbol and type-face specifications and preparing their own warning mechanicals, if they desire.

As a preliminary matter, several factors should be understood. First, the major cost to the cigarette manufacturers is in changing the warning and the frequency of this change. The overall number of warning messages - whether this number is more or less - does not in itself affect the cost. Thus, with the exception of the cost of producing the additional artwork, the cost of the rotational system we recommend is the same whether 6 or 16 warnings are required.

In addition, the greatest cost would be incurred in changing from one symbol format to another; the cost of changing a warning message within the same symbol format is minimal. The reason for this is that changing the symbol formats (i.e. size, shape) entails "ad retouching" production costs, whereas merely changing the black and white type area within a set symbol format is easily accomplished at minimal cost.

This latter factor is the reason we recommend that the implementation of the rotational warning system be coordinated with the introduction of "new" advertising by the cigarette manufacturers and why we recommend the use of only one symbol. This would avoid having the manufacturers incur higher costs of changing the current warning symbol format to the new one.

It is likely, however, that even with some lead time, not all advertising on the date the system goes into operation would be new. The initial implementation of a rotational warning plan would probably require some existing advertising to change to the new symbol. Delaying the date on which the plan goes into effect, and allowing the cigarette manufacturers to include their first new warning in the new symbol in the interim, would lower the costs of the changeover considerably. It is also important to note that the costs of converting to the new symbol would only be incurred once. Thereafter, the only increased production costs of the system would occur when a single advertisement ran for longer than one quarter and the warning message would have to be replaced. This is considerably less expensive than changing the symbol.

A Background: Media Costs

In examining the costs of the rotational warning plan that we recommend, the overall dollar dimensions of the cigarette advertising industry should be kept in mind. For example, the standard industry source for advertising expenditures, Leading National Advertisers, Inc., reports the following for the year 1980:

	Top Ten Brands In Media Expenditures	1980 Total ($000)
1)	Salem	36,978
2)	Winston	36,846
3)	Vantage	32,493
4)	Marlboro	31,349
5)	Kent	29,565
6)	Kool	28,285
7)	Merit	26,500
8)	Now	26,129
9)	Benson & Hedges	24,177
10)	Camel	21,422
	Total	293,744
	All Others	179,891
	GRAND TOTAL	473,635

These figures are the estimated total mounts for magazines, nationally syndicated newspaper supplements (e.g. Parade, Family Weekly) and outdoor only during 1980. They do not include such major media expenditures as regular daily and Sunday newspapers, local newspaper supplements (except the New York Times "Magazine"), outdoor in markets of less than 100,000 population, transit and sidewalk posters, point-of-purchase material or sales promotion materials. Marketing and Media Decisions estimates that the industry spent $1 billion promoting cigarettes in 1980.

These figures are only what it costs to place the advertising. The production costs can average 10% of media costs.

In short, hundreds of millions of dollars are spent each year on cigarette advertising; tens of millions more are spent creating and producing it. Thus, the production and administrative costs described below can be seen to be quite small in the context of the total costs of cigarette advertising.

For additional background and to further indicate the relationship between media space costs and the costs of making changes in the actual advertisement itself, representative costs for each of the media discussed in this report (except point-of-purchase and promotional) are included below:

1. **Newspapers and Newspaper Supplements**

Representative Daily Newspapers	Circ. (000)	Media Costs ½ Page B/W	Media Costs Page B/W
New York Times	914	$10,530	$18,720
Los Angeles Times	1,624	7,392	12,912
Chicago Tribune	790	9,151	14,137
Philadelphia Inquirer	425	5,613	11,227
Boston Globe	492	5,643	10,722
Washington Post	601	8,694	17,381

Representative Sunday Magazine Sections (Supplements)		½ Page 4-Color	Page 4-Color
N.Y. Times "Magazine"	1,477	$10,740	$16,525
L.A. Times "Home"	1,276	10,460	15,900
Chicago Tribune "Magazine"	1,146	7,962	13,117
Phila. Inquirer "Today"	837	6,340	10,105
Boston Globe "Roto."	711	4,692	7,835
Washington Post "Magazine"	828	4,790	8,120

2. Magazines. Below are representative media costs for a random selection of magazines.

Magazines	Circ. (000)	½ Page 4-Color	Page 4-Color
TV Guide	18,871	$38,225	$69,500
Time	4,452	45,880	76,960
Newsweek	2,953	32,330	51,730
People	2,309	23,145	33,100
Sports Illustrated	2,343	29,285	45,050
Cosmopolitan	2,813	16,460	25,315
Playboy	5,747	29,655	50,645
Outdoor Life	1,714	12,650	21,400

3. Transit Posters. Representative transit poster media costs are shown below.

"EXTERIOR" TRANSIT ADVERTISING

City	Size of Poster	# 100 Showing	Cost Per Month
New York	30 x 144	800 units	$65,900
Chicago	30 x 144	500 units	40,000
Los Angeles	30 x 144	600 units	46,200
Philadelphia	30 x 144	400 units	32,120
Washington, D.C.	16½ x 59½	360 units	21,600
Boston	30 x 144	400 units	32,120
San Francisco	30 x 144	300 units	24,090
Detroit	30 x 144	180 units	11,250

4. Billboards and Outdoor Painted Displays. Representative media costs for billboards are shown below. As noted earlier, outdoor painted displays are usually bought for a period of one year and each sign's cost is negotiated individually.

30 SHEET BOARDS

OUTDOOR # "100" SHOWING

City	Population	Non-Illuminated	Illuminated	Total No.of Boards Req.	Cost/ Month
New York	7,179,000	0	255	255	$ 96,645
Los Angeles	9,639,000	68	432	500	145,700
Chicago	6,408,000	56	272	328	94,928
Philadelphia	3,295,000	80	200	280	90,600
Washington, D.C.	3,275,000	12	40	52	15,400
Boston	4,558,000	110	220	330	83,600
San Francisco	4,524,000	44	200	244	70,088
Detroit	4,110,000	0	188	188	62,040

B. Production Costs

In the following we will show the additional production costs that the cigarette manufacturers would incur in handling the health warning message in each of the media described in this report.

In the case of the print media (Newspapers, Newspaper Supplements, magazines) we show two sets of costs, one for "First Publication" and one for "Each Additional Publication." This is necessary because there is a production cost of changing the basic reproduction material which can be used in one publication, and there is also the cost of preparing a set of duplicate reproduction materials for each additional publication where the revised advertisement may be scheduled. As explained earlier, each publication which is scheduled to carry a cigarette ad has to receive a copy of the production material.

As also noted above, the most expensive cost, that of replacing the current warning with a new symbol, will be incurred at most once, and if sufficient lead time is given, this cost may be avoided entirely.

	First Publication	Each Additional Publication
1. Newspapers		
a) Replacing the current warning with a new symbol and new warning message in existing black and white ads.		
i. Offset materials	$650.00	$40.00
b) Changing the warning message within the symbol, after the symbol is part of a black and white ad.		
i. Offset materials	150.00	40.00
c) Including the new symbol, and first warning message in new ads	No additional cost	
2. Newspaper Supplements		
a) Replacing the current warning message with a new symbol and new warning message in existing four-color ads.		
i. Offset materials	650.00	40.00
ii. Rotogravure materials	800.00	60.00
b) Changing the warning message within the symbol, after symbol is part of a four-color ad.		
i. Offset materials	150.00	40.00
ii. Rotogravure materials	245.00	60.00
c) Including the new symbol and first warning in new ads.	No additional cost	

3. Magazines

Additional production costs for handling a new health warning message in four-color magazine advertising are as follows:

	First Publication	Each Additional Publication
a) Replacing the current warning with new symbol and new warning message in existing four-color ads.		
i. Letterpress plates	$4,500.00	$1,800.00
ii. Offset materials	650.00	40.00
iii. Rotogravure materials	800.00	60.00
b) Changing the warning message within the symbol after the symbol is part of a four-color ad.		
i. Letterpress plates	150.00	40.00
ii. Offset materials	150.00	40.00
iii. Rotogravure materials	245.00	60.00
c) Including the new symbol and the first warning message in new ads.		
i. Letterpress plates	No additional cost	
ii. Offset materials	No additional cost	
iii. Rotogravure materials	No additional cost	

4. Transit Posters

The increased production costs of a rotational warning system on transit and sidewalk posters are minimal. The posters are routinely changed monthly. To change to the new symbol, and to change warnings thereafter, would entail a minor revision in the agency-supplied mechanical, at insignificant cost, and then printing. The next posting would, therefore, incorporate the changes at no additional cost.

5. Point-Of-Purchase and Promotional Items

As the warnings on these items would not rotate under our recommendation, there would be no increased production cost. The date on which the items are ordered would control which warning would be placed on them. When the agency supplies the "mechanicals" and/or individual art elements to the specialty firms for printing/production, the correct warning would be incorporated at no extra cost.

6. Billboards

The major cost would be the 5% of each cigarette manufacturer's outdoor advertising expenditures that would be allocated to separate billboards containing only a warning message. There would also be the production cost of creating and printing these billboards. Balanced against these costs, however, is the fact that, under our recommendation, the remainder of the companies' outdoor expenditures would not be accompanied by any warning.

C. Administrative Costs

In addition to the actual advertising production costs described above, cigarette manufacturers and their advertising agencies will incur some additional administrative costs in handling the rotational warning system. It is likely that the administrative time needed will be very small since the warning implementation is just an additional detail in the preparation and placement of cigarette advertising. Little, if any, additional time would be spent on the warning in the initiation and creation of an ad campaign. The Production Manager at the agency would have to spend some small amount of time selecting and purchasing type and patch material for advertisements scheduled to run longer than one quarter. The Traffic Manager would have to spend some additional time in preparing the "schedule flow charts" indicating where and when which advertisements containing which warning messages would run.

Once the system is in operation, there would be some additional time spent in monitoring the schedules to make sure the proper warning, with the proper artwork, was in the proper ad. There would be very little burden in monitoring the system because minor changes in advertising are quite common and are now handled routinely.

The Art Director and Account Manager at the agency, and the Product Manager at the cigarette company, would spend some small amount of their time in supervising the work of the Production and Traffic Manager and in ensuring that the system was operating correctly.

If an advertisement was not scheduled to run longer than one quarter, these minor administrative costs would be even smaller.

The following figures represent an attempt to quantify these minor administrative costs. In calculating these costs, we have deliberately estimated on the high side. The actual costs are probably much lower. For example, we have calculated an hourly rate based on average industry salary levels plus a generous overhead factor. We have also calculated on the basis of each task taking at least one hour. In actuality, and particularly as the system continues to operate, the time spent on these tasks should be much less than one hour, and the costs would be correspondingly lower.

We have estimated, on the high side, the administrative costs of:

1. Replacing the current warning with a new symbol and warning;
2. Changing the warnings within the new symbol each quarter; and
3. Incorporating the symbol and warning in a brand new advertisement.

The figures represent the total administrative costs associated with each advertisement. If the advertisement in question is a magazine ad, the administrative costs are the same whether it appears in four or forty separate magazines. Most cigarette manufacturers do not have a large number of separate advertisements each quarter.

1. Changing an Existing Advertisement

	Hour(s)	@ Rate	Total Cost
Cigarette Manufacturer			
Product Manager	1	$100	$100
Secretarial	4	25	100
Accounting	1	35	35
			$235
Advertising Agency			
Account Manager	3	75	225
Art Director	1	125	125
Production Manager	1	50	50
Traffic Manager	1	50	50
Secretarial	3	25	75
Accounting	1	35	35
			$560
Total			$795

2. Changing the Warnings within the New Symbol Every Quarter

Cigarette Manufacturer			
Product Manager	1	$100	$100
Secretarial	1	25	25
Accounting	1	35	35
			$160

	Hour(s)	@ Rate	Total Cost
Advertising Agency			
Account Manager	1	$ 75	$ 75
Art Director	1	125	125
Production Manager	1	50	50
Traffic Manager	1	50	50
Secretarial	2	25	50
Accounting	1	35	35
			$385
Total = Cigarette Manufacturer & Agency			$545

3. Incorporating the Symbol and Warning in a Brand New Advertisement

	Hour(s)	@ Rate	Total Cost
Cigarette Manufacturer			
Product Manager	1	$100	$100
Secretarial	1	25	25
Accounting	1	35	35
			$160
Advertising Agency			
Account Manager	2	75	150
Art Director	1	125	125
Production Manager	1	50	50
Traffic Manager	1	50	50
Secretarial	1	25	25
Accounting	1	35	35
			$435
Total - Cigarette Manufacturer & Agency			$595

D. Cost for Preparing New Symbol and Health Warning Messages

The cost for the FTC to prepare the new symbol, set type for each of the sixteen health warnings, prepare mechanicals and supply each cigarette manufacturer with reprints to be used as artwork, would be approximately $1,000.

Analysis of Coverage of Tobacco Hazards in Women's Magazines

ELIZABETH M. WHELAN, MARGARET J. SHERIDAN,
KATHLEEN A. MEISTER, and BEVERLY A. MOSHER

CIGARETTE smoking is the number one cause of cancer in the United States. It is also implicated in many other serious diseases, including emphysema, heart disease, gastric ulcers, and chronic bronchitis. During the 1970s, more than two million people died from smoking-related diseases in the United States alone.

These facts come as no surprise to professionals in the field of public health. But they may not be as well known to the general public. Magazines are an important source of health information for the layman. Yet, as R. C. Smith suggested in a 1978 article in the *Columbia Journalism Review*, coverage of this particular health topic, the hazards of smoking, is very limited in most magazines. The exceptions to this general rule have been a few magazines which do not accept cigarette advertising.

In recent years, there has been a significant increase in the number of women who smoke, especially young women who start smoking in their early adolescent years. This trend is reflected in current cancer statistics, which show an alarming increase in smoking-related cancers, particularly lung cancer, among women. In addition women have special cigarette-related health hazards. Smokers have an earlier menopause than nonsmokers. Oral contraceptive users who smoke face special health risks. And, according to the 1979 Surgeon General's report, *Smoking and Health*, "the risk of spontaneous abortion, of fetal death, and of neonatal death increases directly with increasing levels of maternal smoking during pregnancy." Smokers also give birth to premature infants and low birth-weight infants more frequently than nonsmokers.

The American Council on Science and Health (ACSH) has selected smoking and women's health as a primary focus of concern. The coverage of the hazards of smoking in twelve major women's magazines was reviewed. After this, an attempt was made to solicit the participation of twelve women's magazines in a coordinated antismoking campaign.

MAGAZINE SURVEY

Table 1 lists the numbers of articles on smoking presented in each of twelve magazines during a twelve-year period. Articles which discussed the health effects of smoking are enumerated in the first column. Articles telling how to quit smoking or describing personal experiences of people who quit are listed separately in the second column. Not all of these articles carried an antismoking message. Some, such as *Seventeen*'s "Beating the Cigarette Habit" and *Good Housekeeping*'s "Methods That Have Helped Many People to Stop Smoking" did provide encouragement and helpful information for the prospective quitter. However, other articles described quitting as such an unpleasant and difficult experience that they may have discouraged smokers from attempting it. Two such pieces were titled "Smoking: The Sheer Bathos of Beating the Habit" and "When Your Husband Gives Up Smoking, Leave Town."

TABLE 1

Articles on Smoking in Women's Magazines
(March 1967 – February 1979)

Magazine	*Antismoking articles*	*Articles about quitting*	*Not antismoking*	*Total*
Good Housekeeping[1]	6	5	0	11
Seventeen[1]	2	3	0	5
McCalls[1]	2	0	1	3
Vogue[1]	2	0	0	2
Harper's Bazaar[1]	0	1	1	2
Cosmopolitan[2]	0	1	1	2
Mademoiselle[1]	0	1	0	1
Redbook[1]	0	1	0	1
Family Circle[2]	0	0	0	0
Ms.[1,3]	0	0	0	0
Ladies' Home Journal[1]	0	0	0	0
Woman's Day[2]	0	0	0	0

[1] As cited in the *Reader's Guide to Periodical Literature.*

[2] As listed in the tables of contents of these magazines.

[3] Commenced publication July 1972.

The third column in Table 1 includes articles about smoking which did not carry an antismoking message at all. One such article was a "Smoking Psycho-Test," which discussed the hidden psychological information revealed by the way a woman holds, lights, and smokes her cigarette.

It is clear that one woman's magazine, *Good Housekeeping*, has presented substantially more articles about the hazards of smoking than the others. The articles about quitting in this magazine have also been of the helpful type. *Good Housekeeping* also has a policy of not accepting tobacco advertising.

TABLE 2

Articles on Five Health-Related Topics in
Women's Magazines (March 1967 – February 1979)

Magazine	*Antismoking*	*Nutrition*	*Contraceptives*	*Stress*	*Mental health*
Good Housekeeping[1]	6	8	23	4	4
Seventeen[1]	2	2	2	1	1
McCalls[1]	2	4	17	4	23
Vogue[1]	2	8	7	11	6
Harper's Bazaar[1]	0	3	10	7	2
Cosmopolitan[2]	0	19	14	3	178[4]
Mademoiselle[1]	0	2	8	3	2
Redbook[1]	0	6	9	1	0
Family Circle[2]	0	36	8	7	17
Ms.[1,3]	0	0	8	0	0
Ladies' Home Journal[1]	0	0	7	5	2
Woman's Day[2]	0	15	8	8	23

[1] As cited in the *Reader's Guide to Periodical Literature.*

[2] As listed in the tables of contents of these magazines.

[3] Commenced publication July 1972.

[4] This magazine includes a monthly column on this subject.

Articles on smoking in the other eleven magazines surveyed have been rare. Yet most of these magazines run feature articles on health topics regularly. Table 2 shows the number of articles each magazine ran on smoking and on four other health topics of timely interest during the same twelve-year period. Several of the magazines include monthly health columns in addition to feature articles, and they serve their readers by directing attention to new scientific developments which affect the health of women and their families. Yet, during the twelve years surveyed, while scientific evidence on the hazards of smoking to women, and to expectant mothers in particular, appeared with increasing frequency in professional journals, eight of the magazines did not feature a single article on the hazards of smoking, and four of these eight ran no articles on smoking at all.

In a letter to ACSH, the editor-in-chief of one of the largest-circulation women's magazines pointed out that "non-smokers may turn to a piece that is billed as anti-smoking, while smokers may be turned off by it." Her point is important. If smokers don't read articles that are specifically concerned with the health hazards of smoking, these articles are not as useful as they might be. However, information on the risks of cigarette smoking may also be presented in articles and columns on more general health topics, which would attract both smokers and nonsmokers. But when these were examined, disturbing trends were noted.

In some articles on cancer, heart disease, or general health, the risks of cigarette smoking were presented accurately. In addition, news stories about smoking and health were reported accurately in some magazines' monthly health columns, but the dangers of smoking were rarely given prominence. Often the discussion of cigarettes was placed late in the article, rather than on the eye-catching first page. For instance, in an article titled "How to Protect Your Family from Cancer," the relationship between cigarette smoking and lung cancer was described accurately. However, this section was placed after a lengthier section on methods for early detection of cancer, particularly breast cancer. Another example was a feature on "Lungs," which consisted of several pieces on related topics, all starting in the same two-page spread. There was an article on smoking included in the set, but it was placed in a bottom corner, and only a very short section of it was included on the introductory page.

A major women's magazine recently printed an article called "Are You a High Cancer Risk?" written by one of the authors (E.M.W.) of the present paper. The manuscript, as submitted, discussed lung cancer first, emphasizing the role of cigarette smoking. However, when the article appeared, breast cancer was discussed first, and lung cancer was discussed on the last page. To reach this section, the reader would have had to turn to different sections of the magazine twice. Many casual readers probably never got that far.

In other cases the dangers of smoking have been minimized or ignored in articles where a discussion of this health hazard would have been expected. For instance, one women's magazine printed an article, "Preventing Heart

Disease," in which it was stated that contributing factors to heart disease include overweight, rich diets, physical inactivity, and smoking. But, while the other factors were discussed in extensive detail, there wasn't even one full paragraph devoted to smoking.

An even more disturbing article, entitled "The ABC's of Preventive Medicine," ran through the entire alphabet without discussing cigarette smoking at all. Similarly, a feature called "Seventy-six Ways to Save Your Life" included a variety of fundamental and esoteric tidbits about health and safety, but it did not include a suggestion to quit smoking.

In summary, ACSH's review showed that, with the exception of *Good Housekeeping*, the major women's magazines ran articles on cigarette smoking very rarely. A more informal survey indicated that, in broader articles on smoking-related health topics, the role of cigarette smoking was rarely emphasized and sometimes minimized or ignored.

THE INDEPENDENCE FROM SMOKING CAMPAIGN

In early 1980, ACSH wrote to the editors and publishers of *Cosmopolitan, Family Circle, Glamour, Harper's Bazaar, Ladies' Home Journal, McCalls, Mademoiselle, Ms., Seventeen, Vogue, Woman's Day,* and *Working Woman,* to request their participation in a July 1980 antismoking campaign. The editor of each magazine was asked to include an article on smoking and women's health in the July issue, to focus attention on this subject. The assistance of ACSH scientists in the preparation of these articles was offered, at no fee. Since a similar cooperative effort had been used by many of these magazines before, to focus attention on the Equal Rights Amendment, it was hoped that the response would be positive. Since it was suspected that pressure from tobacco advertisers might discourage a single magazine from running a strong antismoking piece, a coordinated activity was planned, to minimize the risk of lost advertising revenue by any one magazine.

With one exception the reaction of the magazines to this suggestion was negative. Most never replied to ACSH. Those editors who did reply declined to participate. The letter from one editor was extremely hostile to ACSH's suggestion that pressure from the tobacco industry might have influenced that magazine to remain silent on the subject of cigarette smoking and health. The magazine in question had published no articles on smoking during the twelve years covered by the survey.

The only positive response to ACSH's suggestion came from *Seventeen*, which featured an article, "Up in Smoke," in its July 1980 issue. In this piece, Julie Rothbard, a teenage smoker who kicked the habit, told her own story of how being a "quitter" made her feel like a winner. ACSH was especially pleased with this response from a magazine aimed at the young female reader, the most important target for messages about women and smoking.

DISCUSSION

The paucity of reporting on the health effects of smoking in most women's magazines is no accident. ACSH members who write health articles for these magazines have been told repeatedly by editors to stay away from the subject of tobacco. Information on the relationship of smoking to health has been edited out of several pieces submitted by ACSH writers. Most editors contacted directly by ACSH would not comment on the reasons for this editorial policy. But it is likely that women's magazines are subject to the same influences that affect other segments of the print media.

There is substantial evidence that the tobacco industry discourages coverage of the hazards of smoking by the print media. It is significant that the major magazines of various types which do run frequent articles on smoking and health, including *Good Housekeeping*, *Science Digest*, *Science*, *Reader's Digest*, *Consumer Reports*, *The New Yorker*, *Hustler*, and *Washington Monthly*, have one thing in common: they do not accept tobacco advertising.

However, for magazines that do accept it, cigarette advertising is an important source of revenue. In 1977, over $400 million was spent to advertise the nation's twenty best-selling cigarette brands. In 1978 alone, Philip Morris Inc. spent over $236 million. Since the 1971 ban on cigarette advertising on television, the major tobacco distributors have spent larger proportions of their advertising budgets on print media. For example, R. J. Reynolds Industries spent $71.5 million on magazine advertising in 1978, a figure that made it the nation's largest magazine advertiser. Two other tobacco companies were also ranked among the top five.

These figures have important implications for the financial health of the magazines which received this advertising. In some cases cigarette advertising made the difference between profit and loss. Robert Liles, a *Good Housekeeping* editor, told ACSH that tobacco ads helped several magazines to survive the 1973 recession.

Unfortunately, this financial dependence may have had some impact on editorial policy. This impact may be indirect; magazines may avoid the unpleasant aspects of smoking as a voluntary courtesy to a major advertiser. However, some people believe that the relationship is more definite. Peter

N. Georgiades, General Counsel for Action on Smoking and Health, said: "Although it is difficult to tell how direct that relationship is, it is clearly the crassest case of journalistic prostitution one will ever see. Many weekly news magazines give only the most washed out, bleached coverage of cigarettes' effect on human health. 'Sanitized' describes their coverage even in their health, science, and medical columns."

The experience of the magazine *Mother Jones* indicates that Mr. Georgiades' allegation may be accurate. In this case, tobacco companies took direct action against a magazine which ran strong articles on the dangers of smoking while accepting cigarette advertising.

A *Mother Jones* representative told ACSH that the magazine's governing board agreed several years ago, after much debate, to accept cigarette advertisements. They made a conscious effort, however, to avoid squelching stories on the health implications of cigarettes. An article on smoking and health appeared in this magazine in April 1978. According to Adam Hochschild, a *Mother Jones* editor, $18,000 worth of advertising was immediately withdrawn by a major tobacco company. Another article on the dangers of smoking was included in the January 1979 issue. Mr. Hochschild told ACSH that "within two weeks of the article's publication, the two remaining tobacco companies cancelled their existing cigarette ad contracts and made it clear that *Mother Jones* would never get cigarette advertising again." Mr. Hochschild reported that as a result of these withdrawals the magazine has experienced "severe problems from the considerable lost revenue."

This incident suggests that the cigarette industry is indeed using advertising revenues to create a "conspiracy of silence" by America's magazine editors. *Mother Jones'* experience indicates that a fear of direct financial retribution from the tobacco industry is not unwarranted.

The American Council on Science and Health believes that the reluctance of a large segment of the print media to inform the public about the health hazards of smoking is a serious impediment to public health education. This applies especially to women's magazines, which many women rely on as a source of accurate health information. A recent survey showed that upper- and middle-class respondents considered magazines to be a source of health information second in reliability only to their doctors' advice. We hope that by calling attention to the problems that magazines face if they attempt to report accurately on the hazards of cigarette smoking, we can discourage future attempts by the tobacco industry to influence the editorial content of periodicals that carry their advertising. We also want to warn health educators of this gap in the print media's coverage of health topics, so that we will all be aware of the need to publicize in other ways the health hazards of smoking.

The magazines' smoking habit

Magazines that have accepted growing amounts of cigarette advertising have failed to cover tobacco's threat to health

by R. C. SMITH

In the seven years that cigarette advertising has been banned from radio and television, American magazines have enjoyed huge increases in revenue from cigarette advertisements. According to *Advertising Age*, the five major tobacco companies spent more than $62 million on magazine advertising in 1970, the year before the ban; by 1976 they were spending nearly $152 million. During that same period, the proportion of all cigarette advertising expenditures that went to magazines doubled. From 1971 through 1976 the tobacco companies spent more than $706 million on magazine advertising, and 1977 expenditures are sure to bring the total to well over $800 million for the seven years that the broadcast advertising ban has been in effect.

During those same seven years, more than half a million Americans have died of lung cancer. The American Cancer Society's authoritative *Cancer Facts & Figures* estimates that more than 400,000 of those deaths were due to cigarette smoking. The 1978 edition of *Cancer Facts & Figures* adds that in addition to being responsible for an estimated 80 percent of all lung cancer deaths, cigarettes have been "implicated in other diseases, ranging from colds and gastric ulcers to chronic bronchitis, emphysema, heart disease and hazards to unborn children." The A.C.S. concludes that "altogether cancer and other diseases due to smoking cause more than 250,000 premature deaths each year." During the seven years since cigarette ads were taken off the air, A.C.S. estimates suggest, well over a million and a half Americans have died of smoking-related disease.

The Tobacco Institute, which speaks for the tobacco industry, continues to insist that a cause-and-effect relationship between cigarettes and lung cancer, emphysema, or heart disease has yet to be established, and that more research is needed. Nevertheless, most experts in the field now seem to believe that enough is known to identify cigarettes as a major health hazard.

"Practically nothing in medicine is as clear," Daniel Horn, head of the federal National Clearinghouse on Smoking and Health, told *The Washington Star* recently. "It's ridiculous to continue to argue about whether or not smoking is harmful."

In addition to the smoking-related deaths, there are the huge social costs that are consequences of smoking. A recent National Cancer Institute publication estimated that $17 billion annually in medical care, accidents, lost worktime, and lowered productivity could be laid to smoking. (It has been estimated that 10 percent of all medical and health-care costs are smoking-related.)

By anyone's measure, these estimates of the toll in lives and resources claimed by the cigarette-smoking habit ought to recommend the subject to any American magazine that claims to serve its readers by keeping them informed of important social issues. Such simply has not been the case.

A survey of the leading national magazines that might have been expected to report on the subject reveals a striking and disturbing pattern. In magazines that accept cigarette advertising I was unable to find a single article, in seven years of publication, that would

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R. C. Smith is the managing editor of the Review.

have given readers any clear notion of the nature and extent of the medical and social havoc being wreaked by the cigarette-smoking habit. The records of magazines that refuse cigarette ads, or that do not accept advertising at all, were considerably better.

Of all magazines, *Reader's Digest* over the years has provided the most thorough and aggressive coverage of the links between cigarettes and disease. At least since the 1950s the *Digest* has published a steady stream of articles on the subject, most of them medically authoritative, and all but one of them generated by the *Digest* itself (the lone exception came from the *Christian Herald*). The magazine's 1976 output of articles on cigarettes and health provides an impressive example of the *Digest*'s coverage. In January there was "What Smoking Does to Women"; in May, "Cigarettes — and Sudden Death" (subtitled "'Every cigarette smoker should witness an autopsy like this'"); in July, "Time to Crack the Tobacco Lobby!"; in August, "Beware that Cigarette Cough"; and in October and December a two-part series, "Poison Gases in Your Cigarettes," which described the results of a *Digest*-sponsored analysis of the levels of carbon monoxide, hydrogen cyanide, and nitrogen oxides found in cigarettes. The *Digest*'s performance over the years has been unique. No doubt the prose at times was too strong for many journalists (". . . . black lungs sliced open on a cutting board, the brain in a jar of Formalin, are shocking post-mortem exhibits of an appalling indifference to the most serious health problem in this country today"), especially during the early years of the cigarette controversy, when the magazine's coverage was considerably ahead of its time. Nevertheless, the *Digest*'s handling of the medical evidence against smoking has been exemplary.

The only other magazine that has devoted much space to the subject has been *The New Yorker*. It has published a number of long articles by reporter Thomas Whiteside on the subject of the political and advertising strategies employed by the tobacco industry to counter the growing concern of the public and the government over the dangers of cigarette smoking. Much that is known about the shrewd and tireless efforts of the tobacco companies to keep their products before the public has been due to Whiteside's articles. (Last June, in an editorial, *The New Yorker* also criticized magazines, including the *Review*, for continuing to accept cigarette advertising.)

In May 1976, *Consumer Reports*, which accepts no advertising, examined "The Changing World of Cigarettes," noting in one article the tobacco industry's switch to low-tar, low-nicotine cigarettes and, in a second article, describing the great increase in cigarette advertising in magazines and newspapers. The magazine urged Congress to ban all cigarette advertising.

T*he Washington Monthly*, a magazine that does not accept cigarette advertising, has run at least two strong articles on cigarettes. One, published in June 1977, was "How to Make the Tobacco Companies Pay for Cancer," by Seth Kupferberg. The other was "The Cigarette Scandal" (February 1976), written by contributing editor James Fallows, who is now President Carter's chief speechwriter. Fallows summarized the medical evidence against smoking, traced the anomalies of the government's tobacco policies, and concluded with a denunciation of publications that justify accepting cigarette advertisements on free-speech grounds.

The records of national magazines that accept cigarette advertising can only be called dismal. A few have published how-to-quit articles from time to time (itself an admirable thing to do, to be sure), although even those are comparatively rare. But anyone who depended on those magazines for reporting on the subject of cigarettes as a major public-health problem would have found nothing at all in many magazines, and only glancing references in others, primarily the newsmagazines.

The so-called women's service magazines, with the exception of *Good Housekeeping*, which does not accept cigarette ads, have not done their readers the service of telling them about what cigarettes contribute to the ill-health of our society. No full-length articles appeared in *Ladies' Home Journal*, nor in *Cosmopolitan*. Not even *Ms.*, during its six years of publication, has done anything substantial with the subject. Thus, readers of *Ms.* may not know of their progress toward one kind of equality they perhaps could do without: the lung cancer death rate for women is climbing steadily, and threatens to equal that of men — largely, health officials say, because American women began smoking decades later than men and usually smoked less. An editor at *Ms.* quite frankly linked *Ms.*'s failure to publish anything about cigarettes and health to the fact that the magazine is "heavily dependent on cigarette advertising." She added, with some irony, that *Ms.* had rejected an ad for Virginia Slims cigarettes ("You've come a long way, baby") — because it was sexist.

The affluent men's magazines, always generously endowed with cigarette ads, also have avoided the subject. *Penthouse* has published no articles on the consequences of smoking. Nor has *Playboy* — its editors thinking, perhaps, that such articles might not be welcome to readers whose "lust is for life."

The most curious performances of all are those of the two major newsmagazines, *Time* and *Newsweek*. (*U.S. News & World Report* has been interested in only one aspect of the tobacco story: cigarette sales.) While both have reported the individual new pieces of evidence of the ill effects of cigarette smoking on health, neither magazine, in the seven-year period, has published anything resembling a comprehensive account of the subject. (Both magazines carry an average of six to eight pages of cigarette advertisements in each issue.)

It was not because the news they cover each week gave them no news "peg" on which to hang such an account. In fact, both magazines were given a perfect opportunity in January 1976, when a book with the unlovely title *Persons at High Risk of Cancer* was published under the auspices of the National Cancer Institute and the American Cancer Society. The book prompted *Newsweek* to do a cover story, in its January 26, 1976 issue, entitled "What Causes Cancer?"

One would expect that cigarettes, as the leading single cause of cancer in the environment, would be identified as such somewhere in the six-page article.

They were not so identified. In fact, the oddest feature of the article, in which a scientist is quoted as saying that the U.S. faces "a major epidemic" of cancer, was the absence of any estimates at all of the nature and extent of the epidemic. To its credit, the story did mention, in several passing references, that cigarettes are carcinogenic. This, on the third page of the article, was the strongest reference:

> The outcry that follows each successive new disclosure of a possible carcinogen on the dinner table or in the work shop tends to obscure the fact that 60 million Americans continue to expose themselves to tobacco — the least disputed carcinogen of all.

No one, in *Newsweek*'s account, dies of cancer; people are merely "exposed" to a "hazard." Thus, the article overlooked estimates in *Persons at High Risk of Cancer* that of 99,500 *preventable* cancer deaths each year, 80,000 were linked to cigarette smoking. Moreover, *Newsweek*'s chart of the "Top Ten Suspects" in causing cancer listed the ten suspected carcinogens in alphabetical order, with no estimates of their relative importance, and with tobacco appearing ninth, after substances such as arsenic, benzene, and benzidine.

(The June 1976 *Harper's* magazine did a somewhat better job with numbers in a sixteen-page section entitled "The Anti-Social Cell: An Inquiry Into the Nature of Cancer." Its table of "The Most Common Cancers" cited the A.C.S.'s estimates of 84,000 U.S. deaths from lung cancer in 1976; the estimate that "possibly 80 percent of lung cancer would be prevented if cigarette smoking were stopped"; and the fact that smokers also run a higher risk of cancer of the larynx, oral cavity, bladder, and pancreas. The section contained a few other brief references to the link between cigarettes and cancer. The information was sketchy, but it was the best I found in a magazine with cigarette advertising.)

A week after *Newsweek* ran its cover story, *Time* gave two columns of its "Medicine" section to the subject. It disposed of cigarette smoking this way:

> While such personal habits as smoking and drinking alcohol have long been linked with cancer, the researchers noted an intriguing new finding: for people who both drink and smoke, the risk of cancer appears to rise proportionately higher than for those who do only one of these things.

Then it was on to the next article, "Fighting Frostbite."

If the newsmagazines avoided full coverage of the effects on health of cigarette smoking even when the "news" at hand would seem to demand it, then it should not be surprising that both magazines avoided similar coverage on the other occasions when they wrote about cigarette smoking. *Time*'s most ambitious effort was just three weeks before its brief story chasing *Newsweek*'s cover story. A "Time Essay" by Michael Demarest entitled "Smoking: Fighting Fire with Ire" chronicled the attempts by nonsmokers to curb public smoking and strongly implied that such efforts smacked of old-maidism and vigilantism. The essay concluded:

> Indeed, the great mass of smokers might be well advised to organize in defense of their own "civil rights." They might call their league Smokers United to Avoid Vigilante Excesses, the acronym, of course, being SUAVE.

Why have no thorough accounts of the destructive role of cigarettes in our society appeared in American magazines that accept cigarette advertising?

Not all the possible explanations are especially ominous. Some editors no doubt think of the subject as worn out; they hesitate to lecture or frighten their readers.

Finally, though, it is impossible not to attribute much of the reticence of magazines to the economic realities of the magazine business. Advertisers are free, of course, to withdraw advertising from magazines whose contents they find uncongenial, and there is plenty of evidence that the tobacco companies have not been reluctant to exercise this freedom.

But when, over a period of seven years, the hazards of a virtually useless product that happens also to have killed hundreds of thousands of Americans fail to attract the attention of even a single magazine that publishes ads for that product — when this happens, one must conclude that advertising revenue can indeed silence the editors of American magazines. ■

It can be done

One recent newspaper reporting effort demonstrates that reasonably thorough coverage of the subject by a publication that accepts cigarette ads is possible. Last November *The Washington Star* ran a three-part series on smoking and health by reporter Cristine Russell.

The first part, headlined FINALLY, THE CANCER LEADERS START TO QUIT SMOKING, looked at a side of the smoking controversy that has rarely been covered: Russell reported on the smoking habits of high officials in the Department of Health, Education and Welfare, and especially in its National Cancer Institute. She found a sizable number who smoked cigarettes. The article was accompanied by an "H.E.W. Smoking Scorecard," which identified the present and past smoking habits of forty-five top officials in the department. (H.E.W. Secretary Joseph Califano quit a three-packs-a-day habit two years ago, Russell reported.) At the end of the article Russell was identified as a nonsmoker, and her editor for the series as a smoker for twenty-two years who had "no intention to quit."

Russell's second article (EXPERTS PUT SMOKING'S COST TO NATION IN THE BILLIONS) listed the estimated health and social costs of smoking, writing, in her lead paragraph: "If historical smoking trends continue, one of every six Americans alive today — nearly 38 million people — might die earlier than expected because of smoking."

The third article dealt with recent changes in public and government attitudes toward smoking, changes in the industry itself, and a "laundry list" of "proposals weighed by anti-smoking groups and health organizations."

Russell's series was a model of what enterprise reporting on the subject could be, and it was all the more striking because it was published by a newspaper that has often been called "financially troubled." *R.C.S.*

THE AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS

May 12, 1982

The Honorable Henry A. Waxman
Chairman
Subcommittee on Health and the Environment
2415 Rayburn House Office Building
Washington, DC 20515

Dear Chairman Waxman:

The American College of Obstetricians and Gynecologists takes this opportunity to comment on your efforts to increase public knowledge of the potential and serious health hazards associated with cigarette smoking. Representing over 23,000 practicing obstetricians and gynecologists who care for a significant proportion of the smoking population, the ACOG is very concerned about the harmful effects of smoking on both women and their offspring.

A pregnant woman who smokes 20 cigarettes a day will inhale tobacco smoke upwards of 11,000 times during an average gestation and may spend 10 percent of her waking day smoking. Scientific evidence indicates that smoking in pregnancy increases the risk of fetal death or damage in utero and predisposes the mother to increased risk of pregnancy-related complications. In light of similar findings by the 1982 Surgeon General's Report on Smoking and the FTC staff report that indicates an increasing number of women and teenage girls begin and continue to smoke without adequate knowledge of the risks they bring to themselves and their offspring, the ACOG endorses the concept of rotational label warnings as proposed in H.R. 5653.

Printed warning statements on cigarette packages and advertisements constitute only one aspect of the public health campaign that must be waged if we are to successfully educate the public. Our efforts must also be specifically targeted at the teenage population who are susceptible to peer pressure and the attractive role models so often found in cigarette advertising. In addition, more research and programs are needed to help those who choose to stop smoking to do so before they encounter a serious medical experience that mandates that they quit or face a life-threating debilitating illness.

In short, concerned individuals and groups -- whether in the public or private sector -- have a responsibility to do all that is possible and necessary to successfully reduce the nation's smoking habit and prevent young people from falling victim to the serious, preventable health hazards of smoking.

Sincerely,

Ervin E. Nichols, M.D., FACOG
Director - Practice Activities

TESTIMONY PRESENTED TO THE
HOUSE SUBCOMMITTEE ON
HEALTH AND THE ENVIRONMENT

on

H.R.4957
"COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT"

March 26, 1982

HON. RON PAUL, M.D.
MEMBER OF CONGRESS

The legislation under consideration here--the "Comprehensive Smoking Prevention Education Act," H.R.4957--apparently has a good deal of support in parts of the medical community. As a physician, a member of Congress, and a concerned American citizen, I am submitting this testimony on behalf of those of us on the other side--those who think the federal government should be out of the tobacco business altogether.

It is not unusual for the U.S. government to be on both sides of one issue, as it is in the case of tobacco. Just as the United States Department of Agriculture administers programs that keep food prices high and then distributes food stamps to people who can't afford the high-priced food, the government also subsidizes those who grow tobacco while simultaneously spending millions of dollars trying to convince people not to smoke it.

I think one of the biggest problems with government is that the people who run it--the administrators and the politicians--are too far removed from the American people. I believe if you took a few dozen, or a few hundred, reasonable men and women and asked them what they thought of these contradictory government activities, they'd say the system was crazy. I agree. Yet this kind of craziness is typical of the way government operates.

Let's look at the tobacco programs. The people who defend these programs pretend that they are not subsidies--yet that's exactly what they are. Through a very expensive set of programs administered mainly through the Commodity Credit Corporation (CCC) and the Agricultural Stabilization and Conservation Service (ASCS), the U.S. government (that is, the American taxpayers) provides everything from guaranteed prices to protection from competition to the favored few who are the government's accepted tobacco growers--and then spends millions of dollars to inveigh against the use of tobacco by the public.

(Incidentally, I think it's ironic that the tobacco program is brought to us in part by the Commodity Credit Corporation--the same wonderful agency that gave us the infamous Polish loans.)

The government tobacco bureaucracy provides three basic "services" to tobacco growers: First, it sets "price supports"--and any tobacco that cannot be sold at these above-market rates is taken by the government, with the CCC absorbing the loss. Second, the government prevents any new tobacco farmers from going into business. Thus, no one can compete with the farmers who already have the government's blessing and protection. Third, "farm allotment levels"--production quotas--determine precisely how much tobacco each farmer can grow. This, of course, has the effect of propping prices up still further. But the government claims it needs this system of quotas to protect itself from the losses that would result from its "price supports" if the market were allowed to function freely.

These tobacco programs, besides being an affront to many Americans, are also expensive. The taxpayers have lent over $5 billion to the tobacco growers since the program's inception in the 1930s, and spent almost a billion dollars to subsidize tobacco exports. America has brought many great things to the rest of the world, but our subsidies for tobacco exports are surely not one of our proudest achievements.

Each year, tens of millions of tax dollars are spent to keep the tobacco programs going. Cost-free (to the tobacco farmers, not the taxpayers) "inspection and grading" services ran to $6.2 million in fiscal 1980. Research and extension programs (including research into "safer" kinds of tobacco) used up $8.1 million that same year. We taxpayers spent almost $14 million to administer the tobacco price support program, and $500,000 to provide up-to-date "market information" to the participants in this non-market system.

And, though it would seem utterly unbelievable to any reasonable person, we actually spend about a million tax dollars each year to protect tobacco crops from insects and weeds!

Altogether, annual costs for the tobacco programs are about $32 million--and rising. And now we need a new program, according to several members of the Congress, to tell people that cigarettes are bad for them. We need an "Office on Smoking and Health," as well as an "Interagency Committee on Smoking and Health." We need new federal research on smoking, and lots of new rotating warning labels to put on packages of cigarettes. Clearly, something is terribly wrong here.

I want to state emphatically that I dislike cigarettes as much as anyone. As a physician, I know that smoking can ruin your health or kill you. I happen to believe that people who smoke cigarettes are being foolish, and I wish everyone would quit. But I think we are deluding ourselves if we believe this is the way to convince them.

The fundamental assumption here is that the American people are too stupid to make their own personal decisions, and that the all-knowing and benevolent government must guide them down the right path. When a nudge will not do, we must push them. When a push will not do, we must knock them over with the full weight of a giant government bureaucracy.

Well, I take strong exception to that view of things. I do not believe Americans are incapable of making their own choices--and I think everyone has the right to make those choices, even if they turn out to be wrong. Certainly the federal government is in no position to be delivering any lectures.

Look at the hypocrisy of it all! The American government pays tobacco farmers to grow their crops, props up the prices and guards against the possibility of competition, accepts the losses when such losses occur, and generally treats the production of tobacco as if it were some kind of wonder drug. And now Congress wants a new federal bureaucracy to tell people not to smoke! How much more ludicrous could it be?

It is the growing body of medical evidence that has convinced many smokers to quit--not the federal government. We are today much more aware than at any time in history of the dangers of cigarette use, and many people are taking heed of this new knowledge. But I challenge the idea that the federal government deserves the credit. To the contrary, I believe the government's role has been an utterly contemptible one.

The American people can see the mixed signals coming out of Washington. *The government supports tobacco*--otherwise, why would it insist on continuing the tobacco subsidies? But the government also feels guilty, apparently--so it tries to make amends through "smoking prevention and education" programs. Sadly, many of the same Congressmen who voted for the tobacco programs will also vote for this new "smoking prevention" program.

This is typical--tragically--of the way government works.

I say that government should just get out of the tobacco business. We should stop helping the tobacco farmers grow the cancerous crop, and stop telling the American people what they shouldn't do with it once it's grown.

The American people have more good sense than the government has ever had--only government could create the kind of mess the tobacco programs represent. It is time to acknowledge the good sense of the American people, and to stop trying to tell them how to live their lives. We don't need new government programs, agencies, or laws to solve the problems of cigarette smoking. Instead, we need to do what we should have done long ago--stop encouraging and subsidizing the production of tobacco.

TESTIMONY OF REPRESENTATIVE
AUSTIN J. MURPHY ON H.R. 4957

I appear before you today as a nonsmoker. I have no quarrel with the conclusions in H.R. 4957 that cigarette smoking is a serious public health problem in the United States or with the concept of attempting to reduce the prevelance of smoking in America. I do, however, have grave reservations about the constitutionality of that part of H.R. 4957 which would require additional and varied warnings in cigarette advertising.

I have long had a deep interest in constitutional law. As a result I have been fascinated by the Supreme Court's recent decisions which have extended First Amendment protection to advertising. While all the implications of First Amendment protections for commercial speech are not yet apparent, it is already clear that government discretion in regulating advertising is limited.

Ten years ago the Congress could have placed almost any restriction it wanted on cigarette advertising. It was not until 1976 that the Supreme Court clearly announced that advertising is entitled to the protections of the First Amendment.[1] In 1980 - less than two years ago - the Supreme Court first described with some specificity the standards by which it would review attempts to regulate advertising.[2]

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[1] Virginia State Board of Pharmacy v. Virginia Citizens Consumer Council 425 U.S. 748 (1976).

[2] Central Hudson Gas v. Public Service Commission, 447 U.S. 557 (1980).

This 1980 decision set forth a four part test to judge advertising regulations:

1) Is the advertisement for a lawful activity and not misleading?

2) Is the asserted governmental interest substantial?

3) Does the regulation directly advance the governmental interest?

4) Is the regulation no more restrictive than necessary?[3]

Cigarette smoking, unfortunately, is lawful in this country and H.R. 4957 does not contain a finding that cigarette advertising is misleading. I would agree that protecting the public health is a substantial government interest. My concern with the legislation therefore involves the last two parts of the Supreme Court's "commercial speech" test.

It is my understanding that there is a substantial controversy over whether health warnings in cigarette advertising are effective in reducing the smoking habit. For example, Michael Waterson has told this Subcommittee that his research efforts on behalf of the Advertising Association, which is based in the United Kingdom, have led him to conclude that health warnings alone have no effect on cigarette consumption. It therefore seems to me that the bill's health warning requirements may not be able to meet the constitutional mandate that the health warning requirements directly advance the public health interest asserted by H.R. 4957.

The last requirement of the Supreme Court's test is that the regulation be no more restrictive than necessary. Therefore, if it is found that, contrary to Mr. Waterson's testimony, health warnings are effective in deterring smoking, it must still be determined whether the warning system established by H.R. 4957 is no more restrictive than necessary. All cigarette advertising currently contains a health warning pursuant to consent agreements between the cigarette companies and the Federal Trade Commission. The Federal Trade Commission's staff has said that over 90 per cent of the American public knows the basic message of the current health warning: cigarette smoking is hazardous to health. This fact suggests that the current health warning is understood even if not heeded. Under these circumstances I think there is a substantial constitutional question of whether H.R. 4957's warning requirements are the least restrictive means necessary to achieve the goal of educating the public.

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[3] Id at 566.

The primary question raised by H.R. 4957's advertising requirements is whether the time for this legislation has already come and gone. Ten years ago, before health warnings appeared in cigarette advertising and the Supreme Court extended First Amendment protection to advertising, the consideration of a requirement of health warnings in cigarette advertising would have been a relatively simple matter. Now there already is a health warning in cigarette ads. There is also survey evidence that almost all Americans know that smoking is a health hazard. What we need in addition to the present warning is an educational effort to actually show young people the result of smoking. Additional warnings will not accomplish that.

Last, but not least, the Supreme Court has made it clear that we are not free to impose whatever regulations we wish on advertising. I therefore have serious concerns that the requirement of health warnings in H.R. 4957 may violate the constitution and would be invalidated by the Courts leaving us with no warning at all.

As legislators we have a duty to enact legislation in a responsible manner. I therefore urge reconsideration of H.R. 4957 in light of the Supreme Court's decisions on advertising and the First Amendment.

STATEMENT BY CONGRESSMAN JOHN L. NAPIER TO THE SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENTOF THE COMMITTEE ON ENERGY AND COMMERCE.

Mr. Chairman,

I oppose H.R. 4957 and H.R. 5653 because they threaten farmers, workers, and employers in the tobacco industry with economic hardship and some with catastrophe without any countervailing benefit to the American people.

The Southeastern United States, and particularly my congressional district, depends on the production of tobacco for its economic livelihood. Any unnecessary disruption of the production of this commodity would have severe economic impact on the people I represent.

Tobacco is raised on more than 6,600 farms in my state. Among them they plant more than 55,000 acres of tobacco---fewer than ten acres apiece on the average, but sufficient to financially support the families involved. Tobacco production touches the lives of nearly 34,000 people who earn all or parts of their living from it.

For the district's economy, that labor translates into farm incomes of more than 30 million dollars a year. Sales of tobacco leaf from those farms bring in nearly $170 million. Auction warehousing accounts for $173 million. Distribution of tobacco products accounts for nearly another $170 million; and ultimately results in retail tobacco product sales of a quarter of a billion dollars.

These activities produce more than 36 million dollars every year for our state treasury, and another 54 million in federal tax collections.

The economic contribution of tobacco to the Sixth Congressional District is extremely vital. Nearly one out of every 20 employed persons in my district is in some manner involved in tobacco production. More than ninety percent of the state's tobacco production originates in my district, and all of the auction warehousing in the state takes place there.

The severe economic hardship that would result from this ill-conceived legislation would be loss of export markets and countless American jobs that depend on exports. These bills go far beyond existing legislation by requiring health warning labels on packages of cigarettes produced in this country for export. American cigarettes carrying a health warning label will then be displayed on tobacconists' shelves in other countries alongside cigarettes of foreign manufacture bearing no such label. There is little doubt that the foreign brand would have a sales advantage over the U.S. brand.

Other countries have decided to adopt methods of informing people of the possible health hazzards associated with smoking different from those used in the U.S. Should we adopt this legislation we would, in fact, dictate policy to other countries by requiring our type of warning in the marketplace.

There is no benefit to the American people by such bills which profess "to establish a national program...to inform the public of the dangers of smoking..." but in fact contains no such program. What they would do is provide a statutory basis for an agency that already exists--the Office on Smoking and Health. There is nothing to stop this agency in its present form from proposing to the Congress any program it feels has merit. All that would be achieved by recognizing the agency in a statute is improving the agency's prospects for higher levels of funding and more staff.

If there is a program in these bills, it lies in the requirement of a hodge-podge of labels on cigarette packages. Under the provisions of H.R. 5653 four labels would be required on cigarettes destined for other countries. One is the health warning label required by the country to which the product is being exported. A second is the warning label mandated by the United States. Third, is a label stating tar, nicotine, and carbon monoxide content. And, finally, a label listing chemical ingredients would appear.

Such a system would be confusing and serve to harass the U.S. tobacco industry rather than to serve the public.

A major change which these bills would affect of replacement of the health warning label now required by seven more specific labels which would be rotated.

Mr. Chairman, I want to recall a recent experience you and your Subcommittee have had with health warning labels. In 1979, when the Senate sent to us a bill requiring warning labels on alcoholic beverages, you adopted a cautious course. You rejected this Senate provision and demanded a thorough study of the efficiency of health warning labels by the Department of Health and Human Services and the Department of the Treasury. You advised these agencies not to sacrifice thoroughness for speed, and the study took a full year. You demanded clear evidence that a label would achieve positive results before you could acquiesce to the Senate's proposal.

I have reviewed the results of your request, a document entitled "Report to the President and the Congress on Health Hazards Associated with Alcohol and Methods to Inform the General Public of these Hazards". It was published last November.

The report contains a section on "Warning Statements as a Method of Informing the Public". Please permit me to conclude my testimony by summarizing what it says.

First, it reviews public receptiveness to warning labels. It cautions that communications experts feel the public is already "over warned" by the federal government. That reaction is heightened, it says, in the case of products for which there is no substitute, such as saccharin or cigarettes.

The report finds that messages with a high level of fear are the least effective. Would that description fit, for example, the cigarette label required by H.R. 4957 which says that "Cigarette smoking may cause Death...?"

The two Departments, their report says, gave special attention to the effectiveness of the present cigarette warning label because it has existed for such a long time. They state "...it is impossible to isolate the impact of...the cigarette warning label...small scale studies...indicate that a health warning label by itself is insufficient to change behavior."

The HSS-Treasury study also looked at the experience with the warning label on saccharin products. It should be noted clearly, Mr. Chairman. They report that after the label went on, the diet soft-drink sales went up.

Their report also dealt with a special audience, women of childbearing age. So does H.R. 4957, which would require one warning stating that smoking by pregnant women may result in birth defects or spontaneous abortion. The report noted a possible backfire: With too many warnings, women might just react with a fatalistic view and ignore them all.

Finally, they raised another point which fits squarely into this bill. They noted the wide variation of amounts pf alcohol in different beverages---that's very much like the wide variation of amounts of tar and nicotine in different cigarettes. They predicted that under a rotating warning system people might notice one kind of warning on a high-tar cigarette and simply think it did not apply to another, low-tar cigarette.

There is much more in the report to Congress from HHS and the Treasury. But the crux of it, Mr. Chairman and members of the Subcommittee, is their conclusion about warning labels on alcoholic beverages indicates that the results do not justify the program.

Mr. Chairman, on behalf of the people in my district, I thank you for this opportunity to present this information to the Subcommittee. I would appreciate your including at this point in the record the warning label section from the HHS-Treasury report.

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STATEMENT BY THE HONORABLE DON FUQUA BEFORE THE SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENT OF THE HOUSE COMMITTEE ON ENERGY AND COMMERCE

Mr. Chairman and members of the subcommittee, I wish to thank you for giving me the opportunity to testify. I am opposed to H.R. 5653 -- as the Chairman of the Science and Technology Committee, as the representative in Congress of hundreds of tobacco farm families in the great Second District of Florida, and as a citizen and taxpayer who thinks that this committee and this Congress have more important matters to deal with in these troubled times.

First, let me say that we have very few physicians and scientists in the Congress. I am more conscious of this precisely because I do head this body's Science Committee, and I feel very uncomfortable with the scientific judgements that are being forced upon us by H.R. 5653. Section 2 of this bill stipulates that we, as Members of Congress, make certain "findings" about the so-called health consequences of cigarette smoking. Mr. Chairman, I cannot in good conscience say that I have found these statements to be true, and I doubt very much that the members of this subcommittee can honestly so find, either.

Mr. Chairman, when the Federal Cigarette Labeling and Advertising Act was amended in 1969, the full committee, of which your subcommittee is a part, had 13 days of hearings on smoking and health. I would point out to you that after what the chairman described as the most extensive hearings in one subject area the committee had ever had, its members came to a conclusion as true now as it was then. They said that the pro and con arguments about cigarettes were the same in 1969 as they had been in similar hearings in 1965. The only difference was that the arguments were supported by a larger statistical base.

I would remind you of cigarette hearings of two other years since, in the other chamber. These were before the Senate Commerce Committee Consumer Subcommittee in 1972 and the Senate Labor and Welfare Committee Health Subcommittee in 1976. I don't think I exaggerate when I say that after a variety of evidence presented on alleged health effects of smoking -- by Members of Congress, government health and regulatory agency officials, state officials, people from the voluntary health associations and expert witnesses in statistics, biomedical research, medicine and other sciences -- each of these hearings ended inconclusively. The proposed legislation did not even come to a vote in either subcommittee.

The major reason, I submit, is the realization among members of both chambers, as well as among government health and regulatory officials, that there are too many scientific unknowns in the cigarette controversy. We know no more about the mechanisms and causes of cigarette-related chronic diseases then we did 17 years ago when we first required a warning on each pack of cigarettes. How can we then come up with instant findings about lung cancer, heart disease and emphysema? How can we, as responsible representatives of the citizens of America, vote to impose these findings on a large and important industry in the form of new labels on a popular and completely legal product?

Mr. Chairman, it is not just the scientific uncertainties that I find in H.R. 5653 that disturb me. It is that in passing this bill we will be setting a precedent for making public policy without an adequate base of knowledge. It is time for all parties to this controversy to admit that there is much that is unknown. Only by doing so will we encourage research to reduce the deficit in knowledge and increase our understanding of the enigmas of cancer and chronic disease.

Mr. Chairman, we do not even know what effects the proposed new warning labels will have on the smoking populace. The Congress has imposed a warning that we know has contributed to the 90 percent awareness of the purported dangers of smoking. We do not know the effect of the proposed new ones and I am reminded of a statement not too long ago by a psychologist, Dan Horn, who has appeared before the Commerce Committee several times in his capacity as Director of the old National Clearinghouse on Smoking and Health.

Dr. Horn is retired now and a sometime consultant to various anti-smoking programs here and abroad. But he cautioned an American Cancer Society hearing in Los Angeles in 1977 against promoting a strengthened warning on cigarette packs. He said he didn't view the warning as an "important source of education," and added that if the warning was made too strong it could prove counterproductive. Mr. Chairman, that is from one who has been in the smoking and health field more than 25 years and is a recognized pioneer in education against smoking.

Mr. Chairman, I have mentioned that I represent a tobacco-growing district in Florida. The flue-cured leaf is important to my state. It is essential to my district. All told, tobacco generates more than 80,000 jobs throught out Florida, from the farmer who grows it to the corner store owner who sells residents and tourists their favorite brands. This 80,000 job estimate by the University of Pennsylvania's Wharton Applied Research Center also included those whose jobs exist because of tobacco's commercial activity within the state, because people who work directly in the industry, their bosses and suppliers, spend their paycheck dollars on the goods and services of other industries, from Pensacola to Jacksonville to the shores of Key West.

The jobs generated directly by tobacco provide almost $11 million annually in wages and income. Those generated indirectly account for almost $100 million in wages. All together, tobacco's state tax contribution is more than $270 million yearly. Another $172 million in tobacco-generated taxes flows every year to the federal government, including $91 million in cigarette excise taxes.

Now I do not wish to equate the economics of tobacco in Florida with the possible harm done to even one person because he or she smoked. Some have said that cigarette smoking is responsible for the unusually high lung cancer death rates in Florida's northeast coastal counties. But we don't know if more people smoke and smoke more heavily there than in my hometown of Altha, or in your home county in California. We have been told by the National Cancer Institute that Jacksonville's high lung cancer death rate, the highest for white males in the nation, indicates the city is paying a price for a World War II shipbuilding boom. We have been told by a Florida State University report that it might be due to chemical pollutants which drift down from industrial cities in New York and Illinois and interact with our warm and humid Atlantic Ocean air to affect our citizens' lungs. So, some say cigarettes, some say wartime ship-building and others say industrial pollution invading us from the North. Mr. Chairman, I respectfully submit that until we know more about the causes of the disease cigarettes have been accused of causing, we have already done enough with the Public Health Cigarette Smoking Act of 1969 to warn smokers.

I cannot conclude without urging that the subcommittee turn down this proposed legislation with the admission that we know as little about the claimed health affects of cigarettes as we do about whether a change in the cigarette warning label will have the effect some desire on consumption.

I would close by telling you about an article in the New York Times on Sunday, March 7, headlined "Cancer Experts Lean Toward Steady Vigilance, but Less Alarm, on (the) Environment." The writer had interviewed two eminent Washington-based scientists, one the former top statistician at the National Cancer Institute. These two are worried, the Times reported, that recent trends suggest that industrial chemicals may cause an upsurge in cancer deaths a decade or two from now. They have voiced their concern in scientific conferences and in an article to be published next May. They noted a tremendous rise in production of synthetic organic carcinogens during the 1970's. They associate with that production an apparent increase in men aged 45 to 84 of cancers associated with occupational exposure. They also note that some cancers related to smoking are increasing in various age groups while others are decreasing, suggesting that factors other than smoking are involved in some of these cancers.

"None of these trends," commented the Times, "even, if real, proves that a major increase is on the way, just that vigilance is needed."

"I don't know if we're on the verge of an upsurge related to our industrial society," said the former NCI scientist. "The sky is not falling," said the other. "But the increases look real and we'd better find out what's causing them."

Mr. Chairman and members of the subcommittee, I really don't see that we can spend this much time and effort planning further restrictions for cigarettes while ignoring the many other suspected causes of cancer and other chronic diseases. I appreciate the opportunity to present this testimony to the subcommittee.

Statement by Rep. L. H. Fountain before the
Subcommittee on Health and the Environment
Committee on Energy and Commerce
March 12, 1982

Mr. Chairman, and Members of the Subcommittee, I am grateful for the opportunity to express my views on the proposed "Comprehensive Smoking Prevention Education Act of 1982". Though I find myself in disagreement with many aspects of this legislation, I will address only those "findings" that are directed specifically at women.

More than ever before, the women of this country are working outside the home. In fact, statistics on both the percentage of women who work and the percentage of women in the overall work force are as dramatic as they are enlightening.

One reason for the substantial increase of working women is the desire to have their own careers. Another is economic necessity. There is a real need for two incomes in one family.

And this need should be appreciated, understood and encouraged by the legislators of our great country. Instead women are continually cautioned and warned and frightened about the implications of their newly assumed role.

When they engage in pursuits that have been characterized in the past as purely male, such as smoking, they are advised by officials in government that "Women who smoke like men will die like men". I suggest to my colleagues on this subcommittee that whereas government may have a role in educating its citizens, it should not engage in scare tactics to accomplish this purpose.

And in that regard I have reason to question the findings in H.R. 5653 that pregnant women who smoke have a higher risk for abortion, stillbirths, premature births, and childweight deficiencies. A full and fair hearing on this subject by this committee would have included testimony from witnesses, expert in this specific area of health and medicine, who are prepared to present contrary evidence, based on their own research and on the research of others.

A full and fair hearing on the subject of oral contraceptives and smoking would have included testimony of expert witnesses who testified on this subject not so many months ago before another subcommittee of the House. As the Chairman may recall, the subject of oral contraceptives and smoking was taken up in 1978 at hearings of the Subcommittee on Intergovernmental Relations and Human Resources during the second session of the 95th Congress, when the Chairman was a member of that committee, and a very good member he was. I am attaching a copy of those hearings which I call to the attention of all of the members of this subcommittee, especially, the distinguished Chairman.

As a member of that subcommittee Mr. Waxman participated with me and others in hearings and deliberations, and took that opportunity to question some of the distinguished scientists who had been called as expert witnesses.

One of these witnesses was Dr. Jean Dickinson Gibbons, Professor of Statistics and Mathematics at the University of Alabama. When asked by Mr. Waxman if certain studies justify that women increase their risk of heart disease by smoking while using the pill, Professor Gibbons replied that these studies did not "justify the presumption of risk".

The principal purpose of those hearings was to examine the scientific basis for the Food and Drug Administration's order requiring an antismoking warning in the labeling of oral contraceptive pills. Our subcommittee investigation lent support to the suggestion that the scientific evidence relied upon by the FDA had been inconclusive and of questionable quality, and that the FDA had based its decision wholly on those questionable data.

Indeed, the suspicions of the subcommittee staff were confirmed by internal FDA memoranda which said:

(1) that one could not infer from the data "that the increased risk of myocardial infarction associated with the current use of oral contraceptives is greater among smokers than among non-smokers";

(2) "that there are insufficient data to evaluate effects of individual risk factors for non-fatal myocardial infarction and almost no data for fatal myocardial infarction";

(3) and that at least one bit of scientific evidence was no more than "ruminations in the absence of facts".

Additional studies have been reported in the scientific literature since the 1978 hearings were conducted. And many of the important points made in 1978 by the non-government, expert witnesses concerning the weaknesses of the earlier studies are reiterated in the results of the more recent studies. Because of my enduring interest in this subject, I have asked Professor Gibbons to review a number of the more recent studies and report to my Government Operations Subcommittee on her findings. She was kind enough to comply with my request on such short notice and has sent me a copy of her report, a summary of which, I understand, she was prepared to present, in person, to the subcommittee. I ask that her full written report be included in the record of these hearings. A copy of it is attached to my own statement.

My reading of Professor Gibbons' evaluation of the current literature is that not much has changed since 1978. Confusion and controversy still prevail on the use of oral contraceptives in general and on the alleged effect of cigarette smoking on the users. Two studies reported in the *American Journal of Epidemiology* in 1979 and 1980 suggest an absence of increased risk of heart attacks among women who smoke and take the pill. A third, *believe it or not*, in the *New England Journal of Medicine* in 1981 suggests *a decreased* risk of heart attack among women who smoke and take the pill!

Professor Gibbons' own evaluation of those studies concluded as follows:

"My primary overall conclusion is that Congressional finding (5) in bill H.R. 4957 is at present groundless because the statistical evidence on which it is based is limited, weak, controversial, and subject to severe criticism by impartial experts."

As recently as 1978, the year of my subcommittee's hearings on this subject, Dr. Donald Kennedy, Commissioner of the Food and Drug Administration, was quoted as saying that even if smoking were not a factor, he would not recommend the pill as a form of contraception for members of his own family.

Yet at the very same time a report in *World Health* entitled "How Safe is the Pill", was saying "Millions of women throughout the world find the Pill effective and acceptable; the overwhelming majority experience no ill-health as a result."

This subcommittee should be aware of the fact that the medical and scientific communities have not agreed on the hazards or safety of pill taking and cigarette smoking either individually or in combination. I suggest, therefore, that the Congress cannot and should not presume to find, in H.R. 5653 or anywhere else, that "women who take birth control pills and smoke are more likely to suffer heart attack or stroke than women who don't smoke".

In fact, I know of no evidence to justify the Congress in presuming or concluding that mere cigarette smoking per se is harmful to one's health. Common sense tells us that the effect of cigarette smoking upon anyone depends upon the person smoking and the extent to which such person smokes. There are a number of items, many of them food, which are harmful to some people--in fact to most people if consumed to excess.

Each person in a free society has the responsibility and the right to make his or her own decisions in connection with the consumption of any legal item.

Thank you Mr. Chairman and Members of the Subcommittee for giving me the privilege of presenting my statement in opposition to H.R. 5653.

STATEMENT OF JEAN D. GIBBONS

My name is Jean Dickinson Gibbons. My current position is Professor of Statistics and Chairman of the Applied Statistics Program at the Graduate School of the University of Alabama. I am currently a Fellow of both the American Statistical Association and the International Statistical Institute and a member of the Committee on National Statistics of the National Academy of Sciences.

I received the bachelor's and master's degrees in mathematics from Duke University and the Ph.D. degree in statistics from Virginia Polytechnic Institute and State University. My previous faculty appointments were at the University of Pennsylvania and the University of Cincinnati. I was a senior Fulbright-Hays scholar at the Indian Statistical Institute in 1973.

I was Associate Editor of The American Statistician for eight years, currently act as editorial collaborator on many statistical journals, including The Journal of the American Statistical Association, Biometrics, and Technometrics, and serve as a reviewer for grant proposals to the National Science Foundation. I am a member of several professional societies and have served two terms on the Board of Directors of the American Statistical Association.

My publications include four scholarly books on statistics and over 30 articles in refereed professional and learned journals in my field. I was named Outstanding Scholar in 1981 and Board of Visitors Research Professor in 1974 at the University of Alabama. My current curriculum vita is attached to this statement.

In February of 1978, I was asked to review the reported statistical studies that formed the basis for the Food and Drug Administration's (FDA) decision to include a boxed warning in the patient and physician labeling on oral contraceptive (OC) products which states that cigarette smoking increases the risk of serious cardiovascular side effects for OC users. In October of 1978, at the request of Representative L. H. Fountain, I testified as an expert witness at a House Subcommittee Hearing on the "Quality of Scientific Evidence in FDA Regulatory Decisions (The Adoption of an Antismoking Warning in Oral Contraceptive Pill Labeling)." My conclusion at that time was that the statistical evidence published in the literature about the interactive effects of smoking and oral contraceptives on risk of cardiovascular disease is quite weak because the sample sizes in most studies are extremely small, the results may be subject to significant sampling errors, and the results in some studies are based on convenient but unfounded assumptions. The authors of these papers in many cases pointed out these limitations of their data and deficiencies in their analyses. At that time I suggested that the FDA should run a controlled experiment to obtain sufficient and relevant data on factors such as length of time of OC use; number of years and amount of smoking; genetic, environmental, and psychological characteristics; among others. My prepared testimony and the discussion following at that hearing are a part of the written record.

Bill H.R. 4957 contains a finding that states "(5) women who take birth control pills and smoke are more likely to suffer a heart attack or stroke than women who don't smoke". This finding is similar to the wording on the patient insert warning on boxes of OC which was at issue in my previous testimony. Therefore, when Representative Fountain again contacted me in February of this year on this matter, I examined and reviewed the statistical studies that have been published on this topic since my previous analysis. My current study included over 20 papers, in addition to the 13 papers examined for the previous testimony and the 3 additional papers that appeared in 1978 and were mentioned during the questioning at that hearing. A complete list of references for the papers appearing since 1978 that I have studied is attached to this statement.

This statement includes a brief analysis of each of the relevant studies published since 1978. I have looked carefully and objectively at the data, findings, and conclusions of the authors, and also performed some independent calculations to measure the statistical relationship between smoking and OC use in their data and to check their conclusions. I have critically analyzed those papers in which the authors claimed there is no (or a slight) increased risk of heart attack or stroke for women who smoke and use OC, as well as those papers where the authors claimed that there is a definite and increased risk of heart attack or stroke. The papers which I reviewed but have not included in this written analysis are, in my opinion, either not relevant to the finding stated in H.R. 4957, or do not add any new results of significance, or do not help clarify the situation.

My primary overall conclusion is that Congressional Finding (5) in Bill H.R. 4957 is at present groundless because the statistical evidence on which it is based is limited, weak, controversial, and subject to severe criticism by impartial experts. There are current studies in the literature that, while also subject to criticism, have come to the opposite conclusion

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and indeed claim that there is no interactive effect of OC use and smoking on the occurrence of heart attack or stroke in women. The evidence for either conclusion is limited and weak and subject to serious scientific criticism. In my professional judgment, I believe that the Congress should not in good conscience find that "women who take birth control pills and smoke are more likely to suffer a heart attack or stroke than women who don't smoke" because the scientific evidence is inadequate. Most of the women who will be affected and influenced personally by this finding will not have the scientific background to form their own conclusions and will interpret the finding as truth, when, in fact, it is at best a questionable opinion that has been neither confirmed nor denied by the data in the reported studies. I again strongly urge the Congress to recommend that a controlled study be carried out with a good data base so that the issue can be addressed properly. More research is urgently needed before Congress can make a finding of such public importance.

In support of these general conclusions, I have attached a brief summary of my analyses of (I.) the group of reported studies that claim no increased significant risk of heart attack and/or stroke, and (II.) the group of reported studies that do claim an increased risk for women who smoke and use oral contraceptives. A complete list of references is attached. Additional details are given in the Appendices.

Analysis of Reported Studies

I. Studies which purport to find no (or slight) increased risk of heart attack and/or stroke for women who smoke and use OC.

A. Krueger et al. (1980) report a collaborative case-control study of death from myocardial infarction (MI) in women aged 15-44 for the period January 1974-June 1975 in regions representing the five largest metropolitan areas in the United States. Data on smoking habits and OC use were reported for 163 women who died of MI and 326 control women; these are reasonable sample sizes on which to base a conclusion and these data appear reasonably reliable.

A primary stated conclusion of the authors is "An interactive effect of OC use and smoking on risk of MI, as reported in recent studies in the U.S. of nonfatal MI, was not found . . ." (p. 672). My independent statistical analysis to verify their conclusion of no interactive effect, shown in Appendix A, is based simply on the reported numbers of smokers and OC users among the cases. My three conclusions from this independent analysis are as follows:

1. In the population of 163 women aged 15-44 who died from MI, the factors of cigarette use and OC use show no statistical association.

2. In the population of 44 women aged 15-44 who died from MI and had no predisposing conditions, the factors of cigarette use and OC use show no statistical association.

3. In the population of 119 women aged 15-44 who died from MI and had predisposing conditions, the factors of cigarette use and OC use show no statistical association.

Another primary stated conclusion of the authors is "Smoking and OC use together appeared to be no more of a risk factor for fatal MI than either smoking or OC use separately, compared to a reference group of nonsmokers and non-OC users" (p. 667). The authors based this conclusion on the odds ratios reported in Table 13, p. 666, and I have verified these odds ratios in Appendix A by independent calculations. The odds ratios and 95% confidence limits (from Table 13, p. 666) are as follows:

Nonsmokers, OC users	2.19	(0.60, 7.33)
Smokers, non-OC users	2.15	(1.38, 3.39)
Smokers, OC users	1.84	(0.81, 4.06)

The fact that the confidence interval for smokers and OC users includes 1.0 is statistical evidence that there is not necessarily any increased risk of MI for women who smoke and use OC over those who do neither; the same conclusion applies to women who do not smoke and do use OC. The confidence level for each of these statements is .95. The fact that the odds ratio for smokers and OC users is smaller than the odds ratios for each of the groups (Nonsmokers, OC users) and (Smokers, Non-OC users) in fact implies the opposite of a synergistic effect.

It is unfortunate that the paper does not give information on the joint characteristics of cigarette and OC use among the deceased controls so that the results could be compared for women who died from MI and women who died from other causes. It is also unfortunate that the data given on both cases and controls have no breakdown according to amount of smoking, years of OC use, and age category within the 15-44 years. Surely the duration of OC use, duration of smoking, amount of cigarettes smoked, and age are important factors to consider in determining whether a relationship exists.

B. Slone et al. (1981) report on a case-control study of the rate of nonfatal MI with respect to the characteristics of duration of current and past OC use, three subcategories of age group within 25-44 years, and four categories of smoking status. The only data on rate-ratios provided in the paper that concern smoking status and OC use are as follows (from Table 7, p. 423):

Never Smoked	2.5
Ex-smokers	2.9
1-24 cigarettes/day	1.5
≥ 25 cigarettes/day	1.4

The authors conclude from these findings that "The rate-ratio estimates declined with increasing cigarette smoking, and the trend was statistically significant. This finding is at variance with previously published observations on current use of oral contraceptives" (p. 423). However, the authors warn of possible bias in this study and recommend that the data be interpreted with caution.

C. Maguire et al. (1979) extend the analysis of some previously reported data on a case-control study of four diagnostic categories of thrombosis (including MI). The primary conclusion relevant here is that their analysis indicates "no strong evidence of modification in the relative risk associated with oral contraceptive use by age or smoking for any of the thrombosis diagnoses considered. It is of interest, however, that in all groups except predisposed venous thrombosis the effect modifier coefficients were negative, suggesting a consistent pattern of decreasing estimated relative risk associated with pill use with both smoking and older age" (p. 193).

D. Belsey et al. (1979) use vital statistics from 21 countries on mortality from cardiovascular disease (CVD) for 1962-74, taking the pre-pill period as controls and post-pill period as OC users. (This is a re-examination of some data used by Beral for a 1976 publication in Lancet, but with an additional two years included.) These authors "find the conclusion of increasing mortality from cardiovascular disease associated with increased levels of pill use unsupported by the data" (p. 85). Their analyses fail to show a significant correlation between percent of women smoking and changes in CVD mortality. The authors do point out, however, the inadequacy of using vital statistics as opposed to a case-control study data base.

E. Jick et al. (1978c) report on a case-control study of nonfatal stroke in premenopausal women. They conclude "In our study, as in the report by the CGSS [Collaborative Group for the Study of Stroke in Young Women], cigarette smoking was only weakly associated with stroke in healthy young women" (p. 59). My independent analysis of their data, given in Appendix B, shows that cigarette smoking is not associated with stroke in this group of women. These authors also state that their "results indicate that oral contraceptives markedly increase the risk of stroke in healthy young women" (p. 59). My independent analysis of their data, also given in Appendix B, shows that there is indeed a positive association between OC use and incidence of stroke at the .001 level of significance. It should be pointed out that the sample sizes for this study are quite small, a total of 56 control and 14 case subjects.

F. Petitti et al. (1978b) use the Walnut Creek Data to study OC use, smoking, and other risk factors for venous thromboembolism and conclude "that OCs and smoking have independent effects in increasing the risk of the idiopathic form of the disease" (p. 484). Their conclusions are based on 17 cases without predisposing conditions grouped as follows:

Table: Number of Smokers and OC Users Among the 17 Cases (from Table 4, p. 483).

	Non-OC Users	OC Users	Totals
Nonsmokers	2	4	6
Smokers	6	5	11
Totals	8	9	17

$\chi^2_1 = 0.7 \quad .30 < P < .50$

Statistical Conclusion: Among women who have the disease and no predisposing conditions, the factors of cigarette use and OC use show no association.

The authors also give relative risk estimates for cases versus controls, and the estimates for smokers and OC users are smaller than the estimates for women with only one of these factors.

Table: Relative Risk Estimates for Cases vs. Controls (from Table 4, p. 483)

	Relative Risk	90% Confidence Limits
Among Smokers, OC users	4.7	(1.3-17.6)
Among Nonsmokers, OC users	12.8	(1.8-90.2)
Among OC Users, Smokers	2.3	(0.8- 7.1)
Among Non-OC Users, Smokers	7.6	(1.6-36.2)

Because the sample sizes for cases are so very small and the confidence intervals are so wide, these results are of questionable reliability.

II. Studies which do purport to find a definite and increased risk of heart attack and/or stroke for women who smoke and use OC.

A. Studies criticized in my previous testimony:

(1) Jain (1977) uses the data in Mann et al. (1975) consisting of 63 women under age 45 who had survived an MI and a control group. Among the cases, there were three nonsmokers and 13 smokers who were using OC at the onset of the MI episode. These numbers are too unbalanced and too small to justify any reliable conclusions about the interrelationship of smoking and OC use on MI. Jain concedes in his paper that these mortality data are based on small numbers and may be subject to significant sampling errors; this

caveat should not be ignored. Further, his analysis is based on two unwarranted but convenient assumptions which he justifies making ". . . because the relevant data . . . are not available" (p. 51). This is a non sequitur, and highly unscientific reasoning.

(ii) Beral (1977) uses the Royal College of General Practitioners (RCGP) data but the numbers are still very small, especially the deaths for nonsmokers. She concedes that "These estimates are based on small numbers and are necessarily approximate. Without more data it is not possible to examine the interrelationships of age, smoking, and duration of oral contraceptive use . . ." (p. 730).

(iii) Jick et al. (1978b) report data on 26 women with acute but nonfatal MI and 59 controls and give relative risk estimates for OC users. However, no MI subjects were nonsmokers who did not take oral contraceptives so relative risk estimates for OC users who smoke could not be obtained.

(iv) Petitti and Wingerd (1978) give relative risk factors for subarachnoid hemorrhage (SAH) for women who smoke and use OC. However, this analysis is based on extremely small numbers, a total of 11 women, which includes none who were nonsmokers and non-OC users, and only six who were smokers and OC users. Certainly this is insufficient data to use as a base for claiming an interrelationship.

8. Shapiro et al. (1979) report a study of 234 premenopausal women with MI and 1742 control premenopausal women. The authors give the following age-adjusted rate ratio estimates and 95% confidence intervals of MI for recent OC users (from Table V, p. 745):

Nonsmokers	4.5 (1.4-14.1)
Smoke 1-24/day	1.2 (0.3- 4.4)
Smoke ≥ 25/day	4.3 (2.2- 8.2)

The estimated rate ratio for heavy smokers is about the same as for non-smokers and the confidence interval for nonsmokers completely includes the confidence limits for heavy smokers. Further, the rate ratio estimate for moderate smokers is considerably smaller then either of those for nonsmokers and heavy smokers, and the confidence interval for moderate smokers includes the value 1.0, which shows no significantly greater risk.

The age-adjusted rate-ratio estimates given in Table VI (p. 746), on the other hand, give a very large rate-ratio estimate for women who are heavy smokers. The authors state that the Table VI results were derived from the data shown in Table V, but the results in Tables V and VI are so inconsistent that I do not see how they could have been obtained from the same data base. Further, the confidence intervals in Table VI are extremely wide for all categories of OC use and similarly for non-OC users who are heavy smokers. The confidence limits for moderate smokers who use OC include the value 1.0, a result that is consistent with the Table V results, but this again implies no risk. My independent calculations of rate ratios from the data in Table V without adjusting for age, as shown in Appendix C, are more consistent with Table V ratios than are those given by the authors in Table VI.

Perhaps more important here is the small frequency of women in each group. See Appendix C. These limited sample sizes alone could justify an argument that the results given by Shapiro et al. are not reliable.

C. Jick et al. (1978a) extend an earlier case-control study of nonfatal MI and its relation to OC use and smoking to include a total of 83 case and 154 control subjects. The authors claim a strong positive association between MI and OC use, and between MI and smoking. My independent analyses of their data, shown in Appendix D, confirm these conclusions. However, these authors also state "In both groups there is an extremely strong correlation between smoking and MI. Of the 83 case patients interviewed, 74 (89%) were current smokers. The corresponding figure for the 153 controls is 67 (44%)" (p. 2,549). As this statement shows, it is certainly true that the percentage of smokers is larger in the case group than in the control group, but this fact has nothing to do with correlation; in fact, the percentages have no relevance for the relationship between MI and smoking because the data bases were not random samples of cases and controls. This paper gives no data on the joint frequences of women wit respect to MI, OC use or smoking characteristics.

D. The NIH Report (1981) states "the Walnut Creek data also confirm that OC users who smoke or who are older than 30 years are at somewhat [emphasis added] greater risk of serious side effects, particularly circulatory disorders" (p. 1,071). However, no specific data or results are given to clarify what the degree of greater risk really is estimated to be.

E. Petitti et al. (1979) also use the Walnut Creek data. Table 1 (p. 1,152) shows the relative risks for MI as 2.9 for women who smoke and 2.8 for women who smoke and use OC (note this relative risk is lower), and the lower 90% confidence limit for women who smoke and use OC is 0.8, less than 1.0; these results for MI cases are based on a total of 26 observations, however. Other relevant results in this table are relevant risks for SAH of 5.7 for smokers and 21.9 for women who smoke and use OC, based on a total of 11 observations, and relative risks for other stroke as 4.8 for smokers and 2.0 for women who smoke and use OC (note this relative risk is lower than the relative risk for smokers), based on a total of 23 observations. Table 3 (p. 1,152) gives incidence rates for the combined types of cardiovascular disease for 3 women under age 45 who neither smoke nor use OC, and 8 women under age 45 who both smoke and use OC. All of the aforementioned results or relative risks are highly questionable because of the extremely small numbers of cases in each subgroup (especially that of women under age 45).

In spite of this severe limitation on reliability, the authors' conclusion is that "smoking and OC use appear to act synergistically to increase the risk of subarachnoid hemorrhage, hemorrhagic stroke, and MI" (p. 1,154). In my opinion, their data do not justify this conclusion at all.

F. Layde, Beral, and Kay (1981) use the RCGP data to study the relationship between smoking and OC use in regard to mortality from SAH and from various circulatory diseases. An independent analysis of their data in Tables IV and V (p. 543), shown in Appendix E, implies that the following statistical conclusions are appropriate: In the population of women aged 35-44, there is no association between OC use and smoking for those who died from SAH, nor for those who died from circulatory disease.

The sample sizes here are small (20 deaths from SAH and 65 deaths from circulatory disease). The authors use the 65 deaths from circulatory disease to compute the relative risk and excess risk estimates for Ever-users vs. Controls in Table V (p. 543), separately for each subgroup of age and smoking. The individual numbers of cases for each of these subgroups are extremely small (see Appendix E, Table 14). The authors do not use these actual numbers to compute relative risks and excess risks; rather they use mortality rates per 100,000 women-years and this leads to an impression of much larger sample sizes. Moreover, the authors state "The relative risk [of circulatory disease] for ever-users was greater among smokers than among non-smokers for each age group" (pp. 543-544). The authors fail to point out that the 95% confidence interval of relative risk for nonsmokers is considerably wider than that for smokers for women aged 35-44, and also for women 45 and over; and in fact the interval for nonsmokers includes the interval for smokers in each case, which implies no significant difference between relative risks for smokers and for nonsmokers.

G. Rosenberg et al. (1980) study the effect of OC use on nonfatal MI in the presence and absence of other predisposing risk factors including cigarette smoking. Their relative risk estimates and 95% confidence intervals for MI for women without other predisposing conditions are as follows (From Table 4, p. 63):

Normotensive nonsmokers	2.8	(1.0-7.8)
Normotensive smokers	1.1	(0.5-2.6)

These risk estimates are for current OC users relative to women who had never used OC.

The relative risk for normotensive smokers is smaller than that for normotensive nonsmokers, and the lower confidence limit is 0.5, which implies that the additional risk for smokers using OC is probably nonexistent. And yet the authors claim "The increase in risk attributable to the combined effect of current OC use, cigarette smoking and hypertension was considerably greater than what would be predicted from the sum of the separate effects of these factors" (p. 59).

Each of the risk estimates given in Table 4 (p. 63) is based on a very small number of cases, however. Only 7 cases were OC users and smokers without other predisposing conditions, and only 12 cases were neither smokers nor OC users. Further, the data base is married U.S. female registered nurses, which is hardly representative of all U.S. females.

Appendix A

Analysis of Data from Krueger et al. (1980)

Table 1. Number of Smokers and OC Users Among the 163 Cases (from Table 13, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	42	6	48
Smokers	101	14	115
Totals	143	20	163

$\chi^2_1 = .0033, \quad .90 < P < .95$

The appropriate statistical conclusion from Table 1 is that in the population of women aged 15-44 who died from MI, the factors of Cigarette Use and OC Use show no association.

Table 2. Number of Smokers and OC Users Among the 44 Cases Without Predisposing Conditions (from Table 14, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	6	1	7
Smokers	31	6	37
Totals	37	7	44

$\chi^2_1 = .016, \quad P = .90$

Table 3. Number of Smokers and OC Users Among the 119 Cases with Predisposing Conditions (from Table 14, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	36	5	41
Smokers	70	8	78
Totals	106	13	119

$\chi^2_1 = .1038, \quad .70 < P < .80$

The appropriate statistical conclusions from Tables 2 and 3 are that in the population of women aged 15-44 who died from MI, the factors of Cigarette Use and OC Use show no association, irrespective of whether there are predisposing medical conditions.

The odds ratios associated with OC use and smoking as given in Table 13, p. 666, are simple proportions of cases versus controls in each smoking category relative to the same proportion for nonsmokers, non-OC users, calculated as follows:

Nonsmokers, non-OC users	Reference Category	= 1.0
Nonsmokers, OC users	6/9 ÷ 42/138	= 2.19
Smokers, non-OC users	101/154 ÷ 42/138	= 2.15
Smokers, OC users	14/25 ÷ 42/138	= 1.84

Appendix B

Analysis of Data from Jick et al. (1978c)

Table 4. Number of Cases and Controls Who Smoke or Not

	Control	Stroke	Totals
Nonsmoker	33	7	40
Smoker	23	7	30
Totals	56	14	70

$\chi^2_1 = .36, \quad .50 < p < .70$

Statistical Conclusion: There is no association between incidence of nonfatal stroke and smoking in these women.

Table 5. Number of Cases and Controls Who Use OC or Not

	Control	Stroke	Totals
Non-OC User	49	3	52
OC User	7	11	18
Totals	56	14	70

$\chi^2_1 = 25.60, \quad p < .001$

Statistical Conclusion: There is a significant association between incidence of nonfatal stroke and OC use in these women.

Appendix C

Analysis of Data from Shapiro et al. (1979)

Table 6. Number of Smokers and Recent OC Users Among the 234 Cases and 1,742 Controls without Regard for Age (from Table V, p. 745)

		OC Use	
Smoking Status		**Yes**	**No**
None	(MI	4	34
	(Control	52	754
1-24	(MI	3	79
	(Control	51	566
≥ 25	(MI	22	92
	(Control	32	287

The rate-ratio estimates unadjusted for age are computed as follows:

	OC Use	
Cigarette Smoking	No	Yes
None	1.0 (Reference Category)	4/52 ÷ 34/754 = 1.71
1-24	79/566 ÷ 34/754 = 3.10	3/51 ÷ 34/754 = 1.30
≥ 25	92/287 ÷ 34/754 = 7.11	22/32 ÷ 34/754 = 15.25

Without the age adjustment, the rate-ratio estimates for non-OC users are similar to those given in Table VI, p. 746 (see Table 7, below). However, the estimates for OC users are much lower than those given by the authors. The rate-ratio for moderate smokers who use OC when unadjusted for age is smaller than the corresponding estimate for nonsmokers, which is also true in Table VI, p. 746. However, the rate-ratio estimate for heavy smokers who use OC is 15.25 when unadjusted for age and Table VI, p. 746, gives the figure as 39 (see Table 7 below); the authors' confidence limits do not include my figure of 15.

Table 7. Separate and Combined Effects of OC Use and Cigarette Smoking in Relation to MI: Age-Adjusted Rate-Ratio Estimates (95% Confidence Limits) (from Table VI, p. 746)

Cigarette Smoking	OC Use No	 Yes
None	1.0 (Reference Category)	4.5 (1.4-14.1)
1-24	3.4 (2.2 - 5.1)	3.7 (1.0-13.2)
$\geq$ 25	7.0 (5.2 - 11.5)	39.0 (22-70)

The authors state that Table VI results are "derived from the date displayed in Table V," but this derivation is by no means clear. Further, the confidence limits are extremely wide for all OC use estimates (and even include 1.0 for moderate smokers) and for non-OC users who are heavy smokers. In addition, the numbers of OC users among the case (MI) women are extremely small, as the following table shows (derived from Table V, p. 745):

Cigarette Smoking	 25-29	 30-34	OC Users 35-39	 40-44	 45-49
None	0	0	0	1	3
1-24/day	1	1	1	0	0
$\geq$ 25/day	3	8	3	5	3

These factors imply that the results presented in Table VI are probably not statistically reliable or valid.

Appendix D

Analysis of Data from Jick et al. (1978a)

Table 8. Number of Cases and Controls with No Predisposing Conditions According to OC Use (from p. 2,549)

	Control	MI	Totals
Non-OC User	49	7	56
OC User	14	23	37
Totals	63	30	93

$\chi^2_1 = 25.14, \quad P < .001$

Statistical Conclusion: There is a significant association between incidence of MI and OC use in women with no predisposing conditions.

Table 9. Number of Cases and Controls With Predisposing Conditions According to OC Use (from p. 2,549)

	Control	MI	Totals
Non OC User	14	19	33
OC User	0	3	3
Totals	14	22	36

$\chi^2_1 = 2.08 \quad .10 < P < .20$

Statistical Conclusion: There is a possible association between incidence of MI and OC use in women with predisposing conditions.

Table 10. Number of Cases and Controls with No Predisposing Conditions According to Cigarette Use (from Table 2, p. 2,549)

	Control	MI	Totals
Nonsmoking Now	72	4	76
Smoking	54	45	99
Totals	126	49	175

$\chi^2_1 = 34.45$, $P < .001$

Statistical Conclusion: There is a significant association between incidence of MI and smoking in women with no predisposing conditions.

Table 11. Number of Cases and Controls with Predisposing Conditions According to Cigarette Use (from Table 2, p. 2,549)

	Control	MI	Totals
Nonsmoking Now	14	5	19
Smoking	13	29	42
Totals	27	34	61

$\chi^2_1 = 9.68$, $.001 < P < .01$

Statistical Conclusion: There is a significant association between incidence of MI and smoking in women with no predisposing conditions.

Appendix E

Analysis of Data from Layde et al. (1981)

Table 12. Number of Smokers and OC Users Among the Deaths from SAH (from Table IV, p. 543)

	Deaths from SAH		
	Nonsmokers	Smokers	Totals
Controls (Non-OC Users)	1	5	6
Ever OC Users	2	12	14
Totals	3	17	20

$\chi^2_1 = .0187$, $.90 < P < .95$

Statistical Conclusion: In the population of women aged 35-44 who died from SAH, there is no association between OC use and smoking.

Table 13. Number of Smokers and OC Users Among the Deaths from Circulatory Disease (from Table V, p. 543)

	Deaths from Circulatory Disease		
	Nonsmokers	Smokers	Totals
Controls (Non-OC Users)	4	6	55
Ever OC Users	13	42	10
Totals	17	48	65

$\chi^2_1 = 1.17$, $.20 < P < .30$

Statistical Conclusion: In the population of women aged 35-44 who died from circulatory disease, there is no association between OC use and smoking.

Table 14. Number of Cases for Each Subgroup of Age and Smoking Status

(from Table V, p. 343)

Age	Ever-Users	Non-OC Users
15-25(4)		
Nonsmokers	0	0
Smokers	1	0
25-34		
Nonsmokers	2	1
Smokers	6	1
35-44		
Nonsmokers	7	2
Smokers	18	3
45-		
Nonsmokers	4	1
Smokers	17	2

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STATEMENT
OF
THE AMERICAN COLLEGE OF PHYSICIANS
on
H.R. 5653, the "Comprehensive Smoking Prevention Education Act of 1982"

Mr. Chairman and Members of the Subcommittee:

The American College of Physicians (ACP) is pleased to have this opportunity to provide its comments on H.R. 5653, the "Comprehensive Smoking Prevention Education Act of 1982."

As you know, the College represents over 54,000 doctors of internal medicine, related non-surgical specialists, and physicians-in-training. The ACP membership includes private practitioners delivering primary health care; medical specialists in such fields as gastroenterology, endocrinology, oncology, and cardiology; medical educators; and researchers.

The legislation presently before the Subcommittee seeks to establish a new strategy for educating and providing information to the American public about the hazards of smoking. It would thereby allow members of the public to make more fully informed decisions as to whether they will choose to smoke. The American College of Physicians strongly supports the central purpose of the legislation and believes that such an effort represents an important and appropriate governmental initiative -- an initiative which we believe is clearly in the public health interest of this Nation.

We concur in the scientific finding that cigarette smoking is the single most important preventable cause of illness and premature death in the United States. In particular, the American College of Physicians believes that the most recent report of the Surgeon General, "The Health Consequences of Smoking," both warrants and requires the full attention of health professionals and policy makers concerned with the protection of the public health. We believe the statements that "cigarette smoking is the major single cause of cancer mortality in the United States" and that there "is no single action an individual can take to reduce the risk of cancer more effectively than quitting smoking" lend strong suport to this Subcommittee's efforts to more fully inform the public of the health risks attendant to smoking.

Despite statements of some to the contrary, there is no scientific dispute with regard to the numerous health hazards presented by smoking. Cigarette smoking is a major cause of cancers of the lung, larynx, esophagus, and oral cavity. Smoking is a contributory factor in cancers of the urinary bladder, kidney, and pancreas. It is a major cause of chronic bronchitis and emphysema and a major risk factor for coronary heart disease and arteriosclerotic peripheral vascular disease. Estimated deaths related to smoking are in excess of 300,000 annually. Lung cancer alone accounts for one out of four of all cancer deaths and it is estimated that at least thirty percent of all cancer deaths are attributable to tobacco use.

It is important to emphasize that at this time, despite the advances made in the treatment of certain cancers, the five year survival rate for lung cancer is less than ten percent. Despite advances in our ability to perform early diagnosis and treatment we have not significantly altered this survival rate. The best preventive measure with regard to cancer of the lung is not to smoke -- this means that those who smoke should stop, and that those who do not smoke should not start.

Lastly, important evidence has been accumulated on the effect of maternal smoking on pregnancy outcomes and the well-being of infants, and there is emerging evidence on the adverse impact of smoking on healthy non-smokers. This latter evidence challenges any statement that smoking is solely a matter of individual choice.

The fact that new evidence is being developed on the impact of smoking on health must not diminish the fact that there already exists a substantial body of irrefutable scientific evidence with regard to the health hazards and increased premature mortality associated with smoking. It should be emphasized that there is clear scientific consensus as to the veracity and import of this existing evidence.

In view of the cost to society -- in both human and economic terms -- which smoking represents and in view of the recognized need to be more diligent as a society in our efforts at health promotion and disease prevention, the American College of Physicians believes that every reasonable effort should be made to alert the public to the range and magnitude of the risks associated with smoking. It is imperative that we make every effort to ensure that those who may choose to smoke are fully cognizant of the risks to their health and the health of others which are being incurred by this action. In addition, we would note that the emerging scientific evidence with regard to the addictive properties of tobacco smoking raises new and additional concerns, and that efforts must be made to convey this information to the American public.

As physicians, in particular as specialists in internal medicine and its related subspecialties, we are fully aware of the overwhelming scientific support for the range of specific warnings outlined in the legislation before the Subcommittee. As providers of primary and continuing care to adults, we have a strong interest in preventive health measures and in the mechanisms by which health-related information is conveyed. We support the proposed changes in the present labeling requirements for cigarette packages. As we have stated, given the clear and dramatic health risks associated with smoking, we believe that every reasonable effort should be made to accurately convey to individuals the nature of this health risk.

We believe that the specificity of the warnings outlined in the proposed legislation will help to emphasize the risk which individuals take every time they smoke. We also believe that it is vitally important to convey to smokers the fact that quitting smoking will reduce the risks to their health. We are pleased to see that under the proposed legislation both messages would be presented. Some might argue that the information conveyed by the proposed warnings is too graphic, but we would counter that the scientific evidence underlying these warnings is also quite graphic. We believe that the specificity of the warnings and their prominence on the packaging materials will provide an important adjunct to our efforts as health professionals to counsel patients in maintaining sound health and reducing the risk of preventable disease and illnesss.

Some have argued that there is not sufficient evidence of the "effectiveness" of the proposed system of rotational health warnings. However, we believe that such an argument is without merit in the face of such substantial and clearcut evidence of adverse health effects and premature mortality, and in view of the tremendous social costs associated with smoking. It is our belief that this preponderance of evidence carries with it a responsibility and in fact a duty to warn the public regardless of whether statistics are presently available which indicate that the public will in fact heed this warning.

We also believe that in the interests of sound international health policy -- and in keeping with a longstanding commitment to being cognizant of the health needs of all people -- warnings should appear on all cigarette packages produced and manufactured in this country, irrespective of where they are ultimately offered for sale.

In addition, we support the provisions included in the proposed legislation which would require that nicotine, tar, and carbon monoxide levels be disclosed. We also support efforts, such as those embodied in legislation pending before the Senate, to require that information be provided to the Federal Trade Commission and the Department of Health and Human Services with regard to those chemical additives used in the manufacture of cigarettes and the quantities of such additives.

At the present time it is extremely difficult to fully assess the relative risks of cigarette additives because of this lack of basic information. Those listings of additives which presently exist are not sufficiently specific in terms of the types and quantities of substances actually being used. The presently unknown health effects of cigarette additives must be explored and such an assessment can only be undertaken if the scientific community has access to more complete information. Given our growing national concern with toxic substances and our developing knowledge with regard to the possible synergistic effects of certain substances, we believe that a national compendium of information on cigarette additives should be considered if we expect to be able to assess present and future health risks.

In conclusion, Mr. Chairman, we support efforts to better inform the public of the health hazards, including the increased risk of premature mortality, associated with smoking. As public policy turns increasingly towards emphasizing the importance of individual efforts at disease prevention and health maintenance we believe that it is essential that individuals receive complete information on the risks associated with smoking. It is our belief that the measure before this Subcommittee will substantially enhance our efforts as health professionals to convey vitally important scientific information to the public.

The American College of Physicians is pleased to lend its support to your efforts and is available to respond to any questions which you might have.

Testimony of

THE AMERICAN COLLEGE OF CHEST PHYSICIANS

Submitted by

Thomas L. Petty, M.D., F.C.C.P.
President

I am Dr. Thomas Petty, Professor of Medicine and Anesthesiology, Head of Division of Pulmonary Sciences, University of Colorado Health Sciences Center. I am also the President of the American College of Chest Physicians, a professional medical specialty society of more than 11,000 physicians, scientists, and educators who specialize in diseases of the heart, lungs, and circulatory system. It is in this latter capacity that I express our support for H.R. 5653, "The Comprehensive Smoking Prevention Act of 1982."

As pulmonary and cardiology specialists, we have seen first-hand the significant health problems associated with smoking and recognize that we are in a unique position to influence our patients to forego smoking in the first place and to stop smoking when health conditions so require. Accordingly, as a society committed to post-graduate medical education, we have viewed the education of physicians (who in turn educate their patients) on the smoking problem as one of our highest priorities.

-- In 1968, the College supported in conjunction with the National Clearing House for Smoking and Health, a national forum on office management of smoking. The procedures of this conference were published in CHEST, the official journal of ACCP.

-- ACCP joined the National Cancer Institute in the preparation of a correspondence course on smoking and its relationship to chronic obstructive lung diseases and asbestosis. This course was mailed to tens of thousands of primary care physicians.

-- The national media focused on our 1972 Annual Scientific Assembly, when the College established a policy requiring smoking physician-registrants to sit in a special smoking section in the assembly room. This section is no longer utilized because so few of our national convention participants now smoke. Since 1980, new Fellows of the College have been asked to take a pledge that they will use their offices, clinics, and hospital environments as health centers to discourage smoking.

-- The College is preparing for publication in CHEST a document describing procedures that the physician should utilize in the office, clinic, and hospital to discourage smoking and to identify the pulmonary specialist's role in smoking cessation.

-- The College is preparing a monograph for distribution in October 1982 identifying the responsibility of chest physicians to inquire of all their patients about their smoking habits and to assist patients in smoking cessation.

The impact of cigarette-smoking is well documented. It causes approximately 300,00 deaths annually; this is one out of seven of all deaths in the United States. In 1980 over 80,000 individuals died from smoking-related emphysema and chronic bronchitis; and approximately 200,000 heart attack deaths were attributed to smoking. The Surgeon General's latest report concludes that "cigarette smokers have overall mortality rates substantially greater than those of the non-smokers."

The statistics on the impact of cigarette-smoking are staggering and clearly call for relief in the form of the proposed legislation. Regarding lung cancer, the Surgeon General's report states that:

1. Cigarette smoking is the major cause of lung cancer in the United States.

2. Smokers who consume two or more packs per day have lung cancer mortality rates 15-25 times greater than non-smokers.

3. Cessation of smoking reduces the risk of lung cancer mortality compared to that of the continuant smoker.

4. The economic impact of lung cancer to the U.S. is approximately $4.25 billion per year in lost earnings, hospital costs, and physician fees.

5. Lung cancer is a preventable disease. It is estimated that 85% of lung cancer mortalities could be avoided if individuals had never taken up smoking.

Over one-third of our membership are cardiologists; they see in their offices and clinics the devastating implications of smoking vis-a-vis cardiovascular diseases.

1. Smoking is a major contributor to the occurrence of heart attacks, sudden death and peripheral vascular disease.

2. Smoking doubles a persons' risk of heart attack and heavy smokers are three times as likely to suffer heart attacks and sudden death.

3. Cessation from smoking greatly reduces the risk of cardiovascular disease.

4. The economic costs of cardiovascular disease to the U.S. is approximately $41.8 billion per year in lost earnings

and health costs, both Federal and private.

Other chronic respiratory diseases are also caused or aggravated by the direct inhalation of cigarette smoke, including chronic bronchitis, asthma, emphysema, pleurisy, pneumoconiosis, bronchiectasis and other disorders. Over 16 million Americans suffer from these diseases.

The most salient characteristic of the smoking problem is that not smoking or ceasing smoking is a means of preventing or delaying the onset of the above-mentioned lung and cardiovascular disorders. This Committee has been apprized of numerous studies that demonstrate increased life expectancy in individuals who stop smoking as compared to those who continued to do so. A twenty-year study (British Medical Journal, 2: 1525-1536, 1976) of over 34,000 physicians in Great Britain showed that deaths from chronic obstructive lung diseases decreased by 24% for those who stopped smoking. The death rate from lung cancer for those who continued to smoke was 16 times higher than non-smokers; after 15 years of abstinence by smokers their death rate from lung cancer fell to only twice the rate of non-smokers. Most recently, on American Study (American Review of Respiratory Diseases, 125:144-51, 1982) documents that cigarette smoking in adults is the most patent predictor of obstructive airways disease and its cessation significantly reduces the future development of the diseases. The use of a simple spirogram (pulmonary function test) markedly enhances the ability to predict obstructive airways diseases.

Another study (New England Journal of Medicine, 300: 213-217, 1979), showed that individuals with coronary artery disease who continued to smoke had death rates 3.95 times individuals in the study group of over 4000 men and women who had stopped smoking.

It is not necessary to further recite the plethora of studies demonstrating the problems associated with smoking. Despite the claims of the tobacco industry that there does not yet exist a scientific casual link between cigarette smoking and major cardiopulmonary health problems, the evidence is quite clear. Government intervention in the marketplace is desirable when the problem is one of national scope, when the costs of regulation are clearly outweighed by benefit to society, and when the government can perform a function not assumable by the private sector. A visible governmental commitment -- or recommitment -- to the eradication of smoking is clearly required. We believe that the passage of H.R. 5653 will do much to assist we practitioners in educating the public regarding health risks involved with smoking cigarettes. We would like to focus on each of the major provisions contained in this legislation.

1. The establishment of a permanent Office of Smoking and Health to administer the program is a basic and necessary component of the bill because it provides an independent, nonpolitical Federal focal point for educational activities. The voluntary sector cannot bear the entire responsibility for educating the public on the risks of smoking. The high cost of advertising and the volume and influence of cigarette advertising are major deterrents to voluntary efforts. The establishment of an Interagency Committee on Smoking and Health to coordinate research and educational efforts of the Federal Government and private sectors will help to avoid duplication of research already being conducted by NIH or other Federal agencies. The bill should specify that the NHLBI and NCI be represented on the Interagency Committee.

Smoking and its health problems are too significant to be subject to the economic or political climate. The Office of Management and Budget, in its effort to reduce domestic spending, has tried to zero-fund the Office twice. Thanks to the efforts of Secretary Schweiker and this Committee, the Office has retained its strength and integrity. A Congressional mandate for the Office will assure continuing public education on the hazards of smoking, and will demonstrate Congress' commitment to a healthier America.

2. We also support the provision that would require the rotation of six new warning labels to appear on cigarette packages and in advertising. The Federal Trade Commission recently determined that "current cigarette advertising practices may mislead consumers by omitting material facts about the health risks of smoking". The report indicated that consumers do not know enough about the harmful effects of smoking and often underestimate the risks of suffering from health problems related to smoking. The current warning label is overexposed and too non-specific as to the health hazards of smoking. We feel that disease-specific warning labels tailored to varying populations (such as pregnant women) will more effectively inform consumers about the health problems associated with smoking. The requirement that limits the use of one label on any brand and its advertising to one year will aid in efforts to get more up-to-date information to the public in the future. Varied warnings will also

promote greater dialogue between physicians and their patients regarding smoking.

3. We strongly concur that advertising information and cigarette packages should be required to identify the carbon monoxide yield of cigarette smoke as well as the content of tar and nicotine. Carbon monoxide is one of the most harmful ingredients of cigarette smoke, in that it reduces the blood's ability to carry oxygen to the cells. Carbon monoxide is particularly harmful to pregnant women and may also be a critical factor in coronary heart disease, sudden death, atherosclerosis, and chronic respiratory diseases.

4. We agree that the rotating warning label requirements and the content label requirements should apply to all imported brands of cigarettes and those which are exported from the U.S. In many countries abroad a health warning label on cigarettes is required by local laws. In those countries where it is not required, American products should provide the same types of warnings as those on cigarette packages distributed within the U.S.

5. Increasing the civil penalties for violation of the new labeling requirements outlined in the bill is desirable to better insure compliance by cigarette manufacturers.

In conclusion, we feel that the evidence of the need for a national education effort on the hazards of smoking is overwhelming. Study after study has established the link between smoking and lung cancer, cardiovascular diseases and many other disorders. Consumers do not have all of the facts to make an educated choice between not smoking and smoking. Cigarette manufacturers spend $1.0 billion on advertising their products each year, or 50 times the amount expended on public education. The statutory establishment of an Office on Smoking and Health will insure a continuing national education effort on the dangers of smoking and rotational labelling will maximize exposure of millions of Americans to at least a minimum level of information on smoking's health hazards.

We heartily welcome the expanded Federal role in addressing the smoking menace to this Nation's health. Mr. Chairman, you and your colleagues are to be commended for recognizing that this problem is one of national scope and deserving of a national legislative and administrative focus. We, as practitioners, educators, and researchers, are pleased to have the opportunity to join hands with the Federal Government to help eradicate the human, social, and economic waste resulting from smoking.

STATEMENT

of the

AMERICAN MEDICAL ASSOCIATION

to the

Subcommittee on Health and the Environment

Committee on Energy and Commerce

U.S. House of Representatives

Re: H.R. 4957 - Comprehensive Smoking Prevention Education Act

March 12, 1982

The American Medical Association takes this opportunity to comment on H.R. 4957. The bill states its purpose is to "educate and provide information to the American public to allow them to make informed decisions as to whether or not they should smoke." The bill would accomplish this by replacing the current general health warning found on cigarette packages with six specific health warnings. One of the six specific health warnings would be required on all cigarette packages and in advertisements. The warnings would be rotated among brands so that each brand would use all warnings within a six year period. H.R. 4957 would also require that "tar," nicotine and carbon monoxide levels be disclosed on packages and in advertisements. The labeling requirements would also apply to cigarettes manufactured for export. The penalty for violation of the act would be increased from the current level of $10,000 to $100,000 and any person could bring civil action to enforce the law.

Comments

The United States Surgeon General stated in his recent report, "The Health Consequences of Smoking," that "Cigarette smoking . . . is the chief, single, avoidable cause of death in our society and the most important public health issue of our time."

A decision to smoke should be made with the knowledge that increased health risks are associated with smoking. For this reason the AMA is supportive of efforts to increase public awareness of the hazards.

The AMA has been involved in many efforts to increase the public's knowledge of the consequences of smoking. The AMA receives and answers many requests for smoking information. In 1978 the AMA published "Tobacco and Health," an account of the comprehensive research program conducted by the AMA. A pamphlet called "Smoking: Facts You Should Know," a copy of which is attached, has been widely distributed along with two anti-smoking posters. Physicians are urged to alert smokers to the risks associated with smoking.

Our comments address only the rotational labeling provisions. We find it very disturbing if, as a recent FTC staff report alleges, approximately ten percent of the population still do not know that cigarette smoking is harmful. Believing a more detailed warning would better inform the public of the harm of smoking, the AMA House of Delegates adopted a report encouraging Congress to require a more explicit warning on cigarette packages.

In testifying before the Subcommittee on Health and Scientific Research of the Senate Committee on Human Resources in 1978 on a similar labeling provision contained in S. 3115, (95th Congress) the Disease Prevention and Health Promotion Act of 1978, the AMA endorsed the rotational label warning concept. We recommended at that time that if adopted the rotational labels "should be evaluated after a period of use to see if there has been any substantial difference in the public's awareness of health problems associated with cigarette smoking." We still believe the effectiveness of the labels should be evaluated.

A more explicit warning, while an improvement, will not be a complete solution. The 1980 Surgeon General's Report on Smoking and Women reveals that smoking is increasing among teenagers. The AMA is concerned about teenage smoking and does not believe a change in the warning alone will fully deal with the problem. Educational programs that emphasize the harmful aspects of smoking from a teenager's point of view should be pursued, and the use of "role models" in cigarette advertisements should be eliminated. Issues of concern to an adult, like a decrease in life expectancy, may not influence a teenager's decision to smoke. More information is needed on youth smoking, such as why they begin to smoke, why they quit smoking, and what method is most effective in urging teenagers to quit smoking. Once this information is available efforts to reduce teenage smoking can be better directed.

Even those who are aware of the dangers of smoking may have difficulty quitting because of the addictive qualities of cigarettes. The recent Surgeon General's report shows that up to 50 percent of those who quit smoking on their own will stay off cigarettes. Assistance must be provided to the remaining 50 percent if smoking is to be decreased significantly. The AMA is developing an audiovisual presentation on how to quit smoking that will soon be available for physicians to use in assisting patients desiring to quit smoking. If adopted, a change in the warning may create more awareness of the dangers of smoking, and more smokers will want to stop smoking. Programs to assist those who want to quit smoking will be needed even more in that case.

Conclusion

The AMA supports efforts to increase public awareness of the hazards of smoking. We believe that the best method to decrease smoking is to help people avoid starting the habit. One method to discourage smoking would be a clear indication of the health hazards of smoking. Thus, the AMA supports a more explicit warning on cigarette packages and in advertisements.

AART AMERICAN ASSOCIATION FOR RESPIRATORY THERAPY 1720 Regal Row, P.O. Box 35287, Dallas, TX 75235 (214) 630-3540

STATEMENT ON HR 4957

COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT OF 1981

SUBMITTED BY

JOHN R. WALTON, RRT

PRESIDENT
THE AMERICAN ASSOCIATION FOR RESPIRATORY THERAPY

Mr. Chairman, my name is John R. Walton. I am the Director of Respiratory Therapy at Northwestern Memorial Hospital, Chicago, Illinois. As President of the American Association for Respiratory Therapy, I welcome the opportunity to provide a statement for the record on the bill HR 4957, "Comprehensive Smoking Prevention Education Act of 1981".

The focus of this testimony is on the subject of "passive smoking" or "second hand smoke" defined as the smoke emitted from the end of a cigarette, cigar or pipe (side-stream smoke) and the smoke exhaled by the smoker (mainstream smoke) which comes into contact with others.

Tobacco smoke is a very complex mixture of gases, liquids and particles. There are hundreds of chemical compounds in tobacco and hundreds more are created when tobacco burns. Some of the most toxic and hazardous compounds are: tar, nicotine, carbon monoxide, cadmium, nitrogen dioxide, ammonia, benzene, formaldehyde, hydrogen sulphide and dozens of others. Any one compound alone can assault the body and cause problems. Together these compounds make smoking the greatest social menace and devastating illness and disease producer of our time.

Non-smokers need to be aware of the disturbing fact that side-stream smoke has higher concentrations of noxious compounds than the mainstream smoke, inhaled by the smoker, as reported by the Surgeon General's Report, The American Lung Association and many other researchers. Studies show that side-stream smoke can have twice as much tar and nicotine; three times as much 3-4 benzpyrene (a suspected cancer causing agent); five times as much carbon monoxide (which robs the blood of oxygen); and fifty times as much ammonia, as mainstream smoke does.

According to Dr. F. Schmidt, in his article "Health Risks of Passive Smokers" at least two-thirds of the toxic substances in cigarette smoke are found in the side-stream smoke, posing a significant health risk for passive smokers.[1] He said studies have shown blood nicotine levels in non-smokers exposed to cigarette smoke of up to 20 percent of the levels found in smokers. Dr. Schmidt also said that a single smoked cigarette releases 30 milligrams of tar and if smoked in an enclosed room can render the air unhealthy according to the Air-Quality Index Standards of the Environmental Protection Agency. Further the concentration of nitrosamines, known cancer causing agents, in side-stream smoke exceeds that found in mainstream smoke by up to fifty times.

Carbon monoxide is a colorless, odorless gas created by incomplete combustion. Car Exhausts and tobacco smoke put it into the air. When you inhale carbon monoxide the gas bumps oxygen molecules out of your red blood cells, prevents oxygen from attaching to the cell and forms a new measurable compound called carboxyhemoglobin. As the amount of this compound increases in your blood, the cells of the body become starved for oxygen. A study recently conducted by Doctors J.R. White and H.F. Froeb at the University of California, San Diego, showed that non-smokers who had worked alongside smokers for many years had lung damage comparable to mild smokers (1-10 cigarettes per day). Another experiment done by researchers at the University of Zurich, Switzerland demonstrated that when a non-smoker is in a smoky room for just half an hour, the results can be carbon monoxide concentrations in the individual's blood as if he had smoked one cigarette directly. Carbon monoxide also stays in the bloodstream and is difficult to remove. After three or four hours, half of the excess carbon monoxide is still in the bloodstream.

industry has set its maximum allowable carbon monoxide level at an average level of 50 ppm (parts per million). Industry efforts are being made to reduce that standard to 25 ppm. The Federal Air Quality Standard has set a limitation of a 9 ppm average for outside air concentrations of carbon monoxide. According to The American Lung Association, when an individual smokes a cigarette, the level of carbon monoxide in the air for the immediate vicinity can be raised to as much as 90 ppm. Other compounds from cigarette smoke produce levels far above dangerous limits. Hydrogen cyanide is a poison that attacks respiratory enzymes. Cigarette smoke contains 1600 ppm; long term exposure to levels above 10 ppm is considered dangerous. Nitrogen dioxide is an acutely irritating gas that can damage the lungs. Levels of 5 ppm in the air are considered dangerous; cigarette smoke contains 250 ppm.

A study done in 1980 by James R. White, Ph.D. and Herman F. Froeb, M.D. evaluated the long term effects of passive and voluntary smoking on the small airway functions in 2100 middle aged subjects.[2] The findings showed that regardless of sex, non-smokers chronically exposed to tobacco smoke had lower flow rates of exhaled air than non-smokers not exposed. In addition, values in passive smokers were not significantly different from those in light smokers and those who did not inhale. Non-smokers in smoke free work environments had the best scores on the spirometric tests; passive smokers, smokers who did not inhale, and light smokers scored similarly and significantly lower; and heavy smokers, not surprisingly, scored the lowest.

One of the most recent and most shocking research studies has been published in Japan. Conducted by Takeshi Hirayama, M.D. of Tokyo's National Cancer Center Research Institute, 91,540 non-smoking wives aged 40 and above were followed for 14 years.[3] The study showed that non-smoking women married to heavy smoking men

(20 or more cigarettes a day) were "up to four times as likely to die of lung cancer as women married to non-smokers".

Many other studies have shown that children with a smoking parent or parents have nearly twice as many respiratory related illnesses as compared to those children of non-smoking parents. Still other adverse effects of passive smoking have been discovered:

1. It can hasten the onset of angina in people with heart disease.
2. It has been shown to be one of the most severe problems faced by people with asthma.
3. It can damage the action of cilia, the microscopic hairs that line the airways of the lungs and whose function is to remove inhaled dust particles and bacteria.
4. It may contribute to the development of atherosclerosis (hardening of the arteries) in much the same way as it does in smokers.
5. In healthy, nonallergic people it has been shown to cause eye irritation, sneezing, coughing, sore throat, hoarseness, headache, nausea and in some cases even memory loss, depressive personality changes, double vision and blackouts.

But these perils of passive smoking are nothing new. Indeed, over 200 years ago Samuel Johnson observed, "It is a shocking thing, blowing smoke out of our mouths into other people's mouths, eyes and noses, and having the same thing done to us."

As more and more research studies are completed, greater undeniable facts will be uncovered. The evidence overwhelmingly indicates that second hand smoke is harmful, extremely toxic, carcinogenic and even deadly. The public must be made aware of these hazards for society and individuals to make educated decisions on this subject. Non-smokers have every right to request protection form smokers. Smokers may take chances with their own bodies and health, but they have no license or right to do so with others.

Research and public education must continue and increase markedly if we are to have any hope of reducing the tremendous social expense, irritation, suffering and death from the most preventable cause - cigarette smoking.

Again I pledge the full support of the American Association for Respiratory Therapy on this important issue. We urge swift Congressional action on the bill HR 4957.

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Statement on Behalf of the National Association for Public Health Policy to the House Subcommittee on Health and the Environment on HR5653, the Comprehensive Smoking Prevention Education Act.

Leonard M. Schuman, M.D.

March 15, 1982

Mr. Chairman and Members of the House Subcommittee on Health and the Environment:

This written testimony in behalf of the National Association for Public Health Policy is in support of HR5653. I am Dr. Leonard M. Schuman, Vice President of the Association and Professor and Director of the Division of Epidemiology of the University of Minnesota School of Public Health. I write not only in behalf of that Association but as a professional with twenty-six years of scientific experience in research on health and disease with particular reference to the role of tobacco use, especially cigarette smoking in the production of disease.

I feel particularly qualified in expressing support of HR5653 not only as an epidemiologist long involved in studies of tobacco as an etiologic agent, but as the epidemiologist member of the Advisory Committee on Smoking and Health to the Surgeon General of the Public Health Service which presented its findings to Surgeon General Luther Terry and the public at large on January 11, 1964. Since then I have been a member of the Task Force on Smoking and Health organized by the Surgeon General to assess the specific hazards of the constituents of tobacco smoke and a consultant to the National Clearing House on Smoking and Health in its annual reports on the Health Consequences of Smoking through 1978. In 1979, I prepared the Introduction and Summary of Findings for the 15th Anniversary Report of the Surgeon General (Califano) on Smoking and Health.

I have spent these past 18 years following the smoking research of my colleagues and contributing modestly to the literature in this field. More importantly, I have watched the all-too-slow progress in convincing the general public of the significantly harmful effects of tobacco, particularly cigarette smoking, and have seen mortality from smoking related diseases increasing yearly in the aftermath of the epidemic of smoking among males which began with World War I and among females which began with World War II. The eighteen years have had elements of frustration for me and thousands of other scientists because of grossly unscientific counter-propaganda of the tobacco industry which continues to issue such statements as: "The evidence is only statistical" and "The controversy has not been resolved". This, in the face of ever strengthening data on relationships elicited initially as associations and now with strong evidences of causality.

This, in the face of repeated replications of studies strongly inferring causality as long ago as the 1950's. What these deliberate spreaders of confusion either do not know or will not admit is that even the establishment of a nuclear chain reaction was based upon statistical calculations of neutron capture. The atomic bomb was "only statistical"!

When the 1964 report to the Surgeon General was completed, a judgement of causality was pronounced between cigarette smoking and lung cancer, chronic bronchitis, emphysema, and laryngeal cancer and between pipe smoking and lip cancer. Strong associations were noted between cigarette smoking and death from coronary heart disease, bladder cancer, stroke and esophageal cancer. Smoking males had a 70% excess mortality over non-smoking males. Babies born to smoking mothers were underweight and frequently premature. The report also emphasized that the "overwhelming evidence points to the conclusion that smoking - its beginning, habituation and occasional discontinuance - is to a large extent psychologically and socially determined". The collected evidences were derived from over 6,000 scientific studies published to that time. The Committee concluded then, 18 years ago, that "Cigarette smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action."

By 1979, on the 15th Anniversary of the first Report of the Advisory Committee to the Surgeon General, 30,000 additional contributions to the literature not only verified the earlier findings but extended them to similar hazards for women whose mortality from lung cancer was increasing rapidly and to other diseases of both men and women, particularly such cancers as those of the oral cavity, pancreas and kidney. (Our own recent studies with kidney cancer have added evidence to the relationshp of one of its forms to cigarette smoking among men and women).

Of special significance is the fact that illness, disability and death among smokers is premature morbidity and mortality, counterventing the rationalization that we all have to die eventually and from something. This morbidity and mortality (340,000 deaths in 1981) has burdened our population (and that of many other countries, as well) with a cost for medical care and a loss of precious productivity which we can no longer afford. The figures of such costs, cited by many are, at best, minimal for, while they are being calculated, morbidity and mortality from selected smoking-related diseases are increasing in rates and in absolute numbers with the growth of the population and in expense with the spiralling inflation of medical care costs.

Even this statement says nothing of the impact on the quality of life among middle-and old-aged smokers.

Although it is true that discontinuance of smoking has shown progress in man and to a lesser extent in women, the rate of decline of the percentage of smokers remains unsatisfactory partly because of ignorance of the hazard, disbelief, and the confusion sown by the tobacco industry. Furthermore, teen-age smoking has been on the increase with the hero models set up for them by tobacco advertising and the age of initiation of the habit has lowered. Frustrating has also been the reduced efforts by an emasculated, inadequately funded National Clearing House on Smoking and Health. Your effort Mr. Chairman, in introducing a bill which, in my opinion, is long overdue, is a significant step toward establishment of a structure for concerted action in education of the public in the hazards of smoking, promotion of research in motivation for smokers to quit, of interagency cooperation for mutual support in these efforts and for a partnership with the voluntary agencies in a common cause. Your approach to rotation of warnings of varying content should reduce, if not eliminate, the ennui which seems to have long ago developed among cigarette smokers with a continuing identical inconspicuous warning. As an epidemiologist whose daily task is the search for causes of disease, I can say unequivocally that cigarette smoking is by far the one single factor which itself is either causally responsible or a risk factor for the greatest number of ailments of man. The absence of smoking in the population could reduce overall mortality by 30%. It must be remembered that tobacco use is an adopted habit which can and must be cast aside. Government must take a hand in this serious problem that is undermining the very core of our national being, for without government intervention, guidance and support, chaos and death will continue to prevail. Prevention is both an ethical and social imperative.

The Origins of the Report of the Advisory Committee on Smoking and Health to the Surgeon General

LEONARD M. SCHUMAN

ON November 9, 1962, in a conference room of the National Institutes of Health, a group of ten scientists designated as the Advisory Committee on Smoking and Health to the Surgeon General of the United States Public Health Service met for their first session. This was the beginning of a dedicated effort which was to take more than fourteen months, numerous meetings in Bethesda, Maryland, and thousands of man-hours of inquiry, analyses, and evaluative writings in the homes and workplaces of these scientists. This first meeting, late in 1962, was ushered in by the usual mutual introductions and expositions of the fields of interest and expertise of the committee members and assisting staff, and with the playing of a popular transcription by Bob Newhart on the first importation of the American Indian's *Nicotiana tabacum* into Europe in 1558. The playing of this recording did not in the least detract from the gravity and seriousness of purpose of the Advisory Committee, but rather set the tone, strengthened the members' resolve, and initiated a feeling of camaraderie among them for the grueling months ahead.

It is doubtful that any single member of the committee was initially completely aware of the historical perspectives of the concern for the health effects of the use of tobacco, or of the remote and immediate antecedents of the establishment of the Advisory Committee. Such historical events were, however, vital in the eventual establishment of this unique committee and its culminating achievement, the *Report to the Surgeon General* (1). Although this report was the first American review and judgmental analysis of the effect of tobacco on all aspects of human mortality, morbidity, and specific diseases in addition to lung cancer, concern for such effects on the population's health did not begin with the Advisory

Committee's *Report*. Tobacco use prompted serious concern over its effects on human health almost from the time of its introduction into Europe. Within sixty years, tobacco had become not only a staple agricultural commodity in Virginia but its principal currency, and America's tobacco culture rapidly expanded both societally and agronomically with the burgeoning migrant population. The increase in tobacco use in Europe was so great that it prompted Simonis Paulli to publish his treatise on the abuse of tobacco in 1665 (2).

Reliable data on the use of tobacco in the total U.S. population began to be available in 1880. In the ensuing eighty years, the per-capita consumption of tobacco tripled and its form of use underwent dramatic changes. Prior to World War I, chewing of tobacco was the principal modality of use, but the decade of the 1920s saw cigarette consumption, and particularly the prefabricated type, increase astronomically, with a decline in use of chewing and pipe tobacco. Although cigarette consumption tended to plateau in the 1930s, a precipitous increase occurred during World War II when the wide-scale adoption of the cigarette habit by women was added to the large-scale consumption by our troops here and overseas. These changes in overall consumption and in the form of tobacco use by the sexes had marked influences on our mortality and disease patterns.

Although concern for the health effects of tobacco use had increased over the previous three centuries, it was not until this century that scientific studies of the problem were initiated. There had been clinical impressions and suspicions recorded before adequate methods of investigation were developed. Holland (3) and Soemmerring (4) in the eighteenth century had separately drawn attention to the relationship between lip cancer and tobacco use. The first systematic approach to the establishment of this association was not made until 1920 by Broders (5). In 1928 Lombard and Doering (6) were the first to observe a higher proportion of heavy smokers among cancer patients in general than among controls. Although vital statisticians in 1900 had begun to note increases in lung cancer mortality, it was in the 1930s that selected disease trends, particularly in lung cancer, became so conspicuous that intensive inquiries into their relationship to tobacco use were initiated. This decade, which saw such reports as that by Pearl on the shorter life expectancy among heavy smokers (7) and, in 1939, the initiation of large-scale epidemiologic studies on the relationship to lung cancer, also marked the beginning of intensive inquiries on the chemical composition of tobacco and tobacco smoke and their pathogenic effects through animal experimentation.

In the ensuing ten years, numerous case-control studies on selected diseases such as lung cancer, chronic bronchitis, emphysema, and coronary artery disease were executed. By the early 1950s their reports had appeared in the literature and four of the eight now-famous cohort mortality studies had been launched. A large number of clinical and pathological observations on the effects of tobacco smoke on man had accumulated. Tobacco was rapidly being incriminated as a health hazard to man.

Little wonder, then, that on January 3, 1954, a group of tobacco manufacturers, growers, and warehousemen established the Tobacco Industry Research Committee (later to be called Council for Tobacco Research—USA) to launch a research program on tobacco use and health. This research council was established to counter the possible effects of smoking and health studies by instituting research of its own. The rapidly accumulating results from a growing number of studies on lung cancer by health scientists were all consistent in behalf of a positive association. These findings prompted the then Surgeon General of the U.S. Public Health Service, Dr. Leroy E. Burney, in June of 1956 to promote the establishment of a scientific study group by the National Cancer Institute, the National Heart Institute, the American Cancer Society, and the American Heart Association to assess the data on the smoking problem. The group agreed that a causal relationship between cigarette smoking and lung cancer existed (8), and Surgeon General Burney placed the Service on record on July 12, 1957, that the weight of the evidence indicated such a causal relationship.

Through this period of scientific endeavor and realization of the health effects of tobacco use, no governmental policy in regard to this health hazard existed. The health forces were obviously too weak to grapple with the tobacco subsystems (9), which consisted of the tobacco growers, marketing organizations, cigarette manufacturers, Congressmen representing tobacco constituencies, leading members of two appropriations subcommittees and of two substantial commerce legislative committees in both houses under whose purview tobacco legislation and related appropriations fell, and officials of the Department of Agriculture involved in several tobacco programs including grower subsidies and export promotion. This formidable array constantly kept the health forces, with their extremely small representation in Congress, off balance. The tobacco subsystem or coalition was obviously strengthened by the smokers who then constituted more than 60% of the male and 30% of the female population of the United States. As the scientific evidence against cigarette smoking mounted and the consumer public became more concerned, the tobacco forces countered with more refutations, citing its own research and branding all associations between smoking and disease based on sound epidemiological methods as "only statistical."

That the tobacco interests were indeed concerned over the scientific findings against tobacco is reflected by the vigor with which they prosecuted their public-relations programs. It is probably no coincidence that the official stand which Surgeon General Burney took on July 12, 1957, in regard to smoking and lung cancer was followed by the creation, in January 1958, of the powerful lobbying group representing the tobacco companies, the Tobacco Institute, Inc. A scientific counter-event, the brilliant analysis and defense by Cornfield et al. (10) of the causal relationship between cigarette smoking and lung cancer, was, however, a convincing argument to most of the scientific world. It led to Surgeon General Burney's reiteration of the U.S. Public Health Service position with the stronger statement that smoking, and particularly cigarette smoking, was the *principal factor* in the increased incidence of lung cancer. However, this position did not constitute policy, for it carried no stipulations for intervention and control. The role of the U.S. Public Health Service in the ensuing two to three years remained one of maintenance of this position of causality, the conduct and financial support of investigations of smoking and its relationship to health, and surveillance of worldwide reports on this relationship. A schizophrenia thus existed in the federal government: on the one hand, a concern for human health hazards from smoking, and, on the other, the promotion of tobacco culture through subsidy and export. This schizophrenia was lopsided, however, for the concern for health did not include policies of intervention.

The sphere of action of the health forces was not totally a barren desert. Members of Congress with true humanitarian interests had repeatedly introduced bills to restrict cigarette sales and convey public warnings, with no success. The tobacco subsystem and the industry's lobbying arm were too powerful for the weak and virtually unorganized health groups.

It was not until 1961 that positive policies for amelioration of the smoking problem began to germinate. What apparently was needed was an objective appraisal of the situation, relatively divorced from governmental and industry influence, by a group of unbiased scientists of impeccable reputations and requisite expertises, who would review the data which had accumulated so rapidly, provide the most dispassionate judgments of their meaning, and recommend courses of action by the government if tobacco was incriminated.

A number of fast-moving events encouraged and hastened policies of intervention. On June 1, 1961, the presidents of the American Cancer Society, American Public Health Association, American Heart Association, and National Tuberculosis Association urged President John F. Kennedy to establish a commission for the study of the tobacco problem. On January 4, 1962, representatives of these organizations met with Surgeon General Luther L. Terry and once more urged action. The Surgeon General submitted a proposal to the Secretary of Health, Education, and Welfare calling for an expert advisory committee to assess the then-existing knowledge "and make appropriate recommendations."

In the meantime, pro-health members of Congress were not inactive. Noteworthy was the introduction in March 1962 of a Senate joint resolution (SJR 174) by Senator Maureen Neuberger of Oregon calling for the establishment of a Presidential commission on tobacco and health. The resolution was never brought to vote, a not unexpected fate given the power of the tobacco subsystem. It has been suggested that Senator Neuberger was "seeking a wider audience than Congress" (9). If her intent was to encourage the health forces to petition the President, this came about in a most indirect way.

In April 1962 Senator Neuberger suggested to the chairman of the Federal Trade Commission that cigarette advertisements failing to carry health warnings were deceptive and asked why the FTC could not provide rules against such practice (11). The newly appointed chairman, Paul R. Dixon, provided Senator Neuberger with hope. Although the FTC had no rule-making powers, he strongly felt this was a power the FTC needed for making its regulatory process effective. If the Commission were to issue a rule such as Senator Neuberger was requesting, and which would hold up in the courts, it would require "competent probative evidence including that furnished by the Public Health Service, that a causal relationship exists between cigarette smoking and lung cancer, heart ailments, etc." (11).

In this same month, Surgeon General Terry provided an even more detailed proposal to the Secretary of Health, Education, and Welfare for an advisory group which would reevaluate the Service's position of 1959. He cited a number of important developments including the unfolding of new position-strengthening studies on major adverse health effects, evidences that medical opinion was now very strongly against smoking, the very recent appearance of the *Report* of the Royal College of Physicians of London, which concluded, after three years' appraisal of data, that "cigarette smoking is a cause of lung cancer and bronchitis and probably contributes to the development of coronary heart disease" (12), and, finally, a request from the Federal Trade Commission for guidance on labeling and advertising of tobacco products.

At a May 1962 Presidential press conference, a reporter, cognizant of Senator Neuberger's ill-fated attempt at a resolution for a Presidential commission, asked President Kennedy his opinion of the studies which were rapidly emerging with incriminating data on smoking hazards (13). The President's response was temporizing, and the consultations between the President's office and the Public Health Service that followed led to Surgeon General Terry's announcement, on June 7, 1962, of the planned formation of an expert committee to review all data on smoking and health.

As evidence of democratic action and avoidance of accusations of bias, Surgeon General Terry invited representatives of the American Cancer Society, American College of Chest Physicians, American Heart Association, American Medical Association, the Tobacco Institute, Inc., Food and Drug Administration, National Tuberculosis Association, Federal Trade Commission, and the President's Office of Science and Technology to a meeting on July 27, 1962, to outline the objectives of the scientific advisory committee, set the rules for the selection of its members, and compile lists of candidates. At this meeting it was decided that "an objective assessment of the nature and magnitude of the health hazard [would] be made by an expert scientific advisory committee which would review critically all available data, but would not conduct new research. This committee would produce and submit to the Surgeon General a technical report containing evaluations and conclusions. Recommendations for actions were not to be part of this committee's responsibility. . . . It was recognized that different competencies would be needed [for this purpose]" (1).

The July 27 meeting compiled a list of 150 scientists and physicians representative of a broad range of medical sciences and with the necessary expertise for evaluating the factors and their interactions in the complex relationship between smoking and health. In the month that followed the 150 were screened by all the representatives of the organizations invited to the meeting. Any organization could veto any name on the list for any reason, and anyone who had made a decision publicly on the relationship would not be included. In this way, a fresh approach to the accumulated up-to-date data without preconceived notions or preset biases would be assured. Thus, the committee could not be branded as for or against any special interest. In this way a final list of names was submitted to the Surgeon General and from this list he selected ten persons who agreed to serve on what became known officially as the Surgeon General's Advisory Committee on Smoking and Health. Their names were announced on October 28, 1962. The fields of epidemiology, genetics, internal medicine, organic chemistry, pathology, pharmacology, and statistics were represented by men known for their investigative prowess and integrity.

The committee was unique in ways other than in their unbiased selection by representatives of agencies deeply concerned with all aspects of the problem. Surgeon General Terry had, from the very outset, assured the members of the committee that their work would be executed with full independence in all aspects of its organization and pursuit. He emphasized its freedom of action and freedom to report as it saw fit. Throughout the conduct of the committee's work reassurances to this effect were provided. The committee's desire to conduct its work in its own way and to obtain the best possible advice and cooperation from outside experts as well as its resolve to have the *Report* totally the product of its labors and its own authorship were completely respected. Thus a deep sense of personal responsibility for a national problem pervaded the group.

The facilities of the entire Public Health Service were at its disposal, particularly the Office of the Surgeon General, the National Institutes of Health, the then Bureau of State Services, and the National Center for Health Statistics, which provided the committee with fresh data analyses of the several prospective studies then in progress. The National Library of Medicine, in the lowest sub-basement offices of which the committee and its staff were housed and its files maintained with top security, provided enormous volumes of reprints of relevant articles and other publications up to 1959 that had been encyclopedically reviewed with annotations by Larson, Haag, and Silvette (14), as well as the publications that had appeared in the world literature from 1958 to late 1963. Over 7,000 reprints were evaluated by the committee and its consultants. The work of the 155 consultants utilized by the committee was performed under contract and comprised special meetings with subunits, subcommittees, or individual members of the committee, evaluations of literature, and special papers. Conferences and meetings of such consultants were held in the National Library of Medicine headquarters of the committee or at the members' home institutions. Major cigarette and other tobacco-product manufacturers were invited to submit statements and any other information relevant to the inquiry. Open meetings were held during the earlier fact-gathering phase, but closed meetings with high-security provisions were instituted when analytic evaluations and judgments were to be made and the sections of the report written. One member spent his vacation leave from his institution writing the Cancer section of the final report. The committee met periodically through December 1963 to evaluate and review drafts.

High security was maintained so that no disconcerting leaks would occur —leaks which would not only have provided premature ammunition for attacks on the committee's deliberations, but would have wasted its limited time in rebuttals and repeated expositions of the process of judgmental evaluation. Guesswork among outsiders was rampant and on at least one occasion came very close to the ultimate judgments of the committee.

The final conclusions of the Advisory Committee were based upon systematic evaluations of clinical, pathological, experimental, and epidemiologic evidence. Judgments of causality followed predetermined criteria on the associations between smoking and a disease entity or process. The elucidation, exposition, and application of these criteria were a notable epidemiologic accomplishment of the Advisory Committee, whose members learned from each other and taught each other the rules and scientific precepts of their individual disciplines. No minority report was written. Scientific evaluations were objective, and the *Report* represented conclusions unanimously acceptable to every member of the committee.

The *Report* was published under extreme security and, on the morning of January 11, 1964, copies were provided to reporters for the press, radio, and television for one hour's study before meeting in press conference with the Advisory Committee, the Surgeon General, and staff directors in the State Department Auditorium.

The Surgeon General accepted the Advisory Committee Report in full on behalf of the Public Health Service on January 27.

The Advisory Committee proved to be an influential force in the policy process of government. As a committee it was not a continuing one, for its official life lasted but fifteen months. Its influence, however, through the various wheels it set in motion, continues to be felt in the seventeen years of successive events, favorable for the most part to the health of the public. The *Report to the Surgeon General,* born despite adversity, may have served to weaken the power of the tobacco subsystem to a modest extent, but it enhanced the resolve, unity, and power of the health forces immeasurably more.

Editor's Note: Dr. Schuman was a member of the Advisory Committee on Smoking and Health to the Surgeon General of the United States Public Health Service (1962–64) and the Surgeon General's Task Force on Smoking and Health (1967–68).

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Smoking as a risk factor in longevity Leonard M. Schuman

Gains in longevity

Man's subconscious quest for a measure of immortality continues unabated; yet, paradoxically he jeopardizes his small share in the immortality of his species by his actions. Man strives for an improvement in his longevity, yet contradicts this striving with certain of his habit patterns and environmental exposures. The gains in life expectancy at birth among so-called Western cultures in the 75 years since 1900 are only slightly, if at all, transmitted to the older age groups in our population. These gains are not added to man's longevity at the upper end of his life span. The major contributions to this large increase in life expectancy in the youngest age groups included the decline in infant mortality from 100 deaths per 1000 live births in 1915 to 16.1 in 1975 and the control of communicable diseases in childhood, chiefly respiratory and enteric diseases, by means of immunization and sanitation respectively (US Department of Health, Education, and Welfare 1979a; Section 2, Part A, pp. 2–3). In Table 1(a) and (b) the dramatic gains in

Table 1 Average remaining lifetime in years at specified ages: (a) for whites by sex: 1900–2 and 1975, USA; (b) for non-whites by sex: 1900–2 and 1975, USA

(a) Age	White male			White female		
	1900–2†	1975	Gain	1900–2	1975	Gain
0	48.2	69.4	21.2	51.1	77.2	26.1
1	54.6	69.6	15.0	56.4	77.1	20.7
10	50.6	60.9	10.3	52.2	68.4	16.2
20	42.2	51.4	9.2	43.8	58.6	14.8
30	34.9	42.2	7.3	36.4	49.0	12.6
40	27.7	33.0	5.3	29.2	39.4	10.2
50	20.8	24.3	3.5	21.9	30.3	8.4
60	14.4	16.8	2.4	15.2	21.9	6.7
70	9.0	10.9	1.9	9.6	14.4	4.8
80	5.1	6.7	1.6	5.5	8.6	3.1

(b) Age	Non-white male			Non-white female		
	1900–2†	1975	Gain	1900–2	1975	Gain
0	32.5	63.6	31.1	35.0	72.3	37.3
1	42.5	64.4	21.9	43.5	73.0	29.5
10	41.9	55.8	13.9	43.0	64.4	21.4
20	35.1	46.3	11.2	36.9	54.7	17.8
30	29.3	38.0	8.7	30.7	45.3	14.6
40	23.1	29.8	6.7	24.4	36.2	11.8
50	17.3	22.4	5.1	18.7	27.9	9.2
60	12.6	16.3	3.7	13.6	20.7	7.1
70	8.3	11.3	3.0	9.6	14.4	4.8
80	5.1	8.5	3.4	6.5	11.0	4.5

†10 states and D.C. in 1900–2; entire U.S. in 1975.
Source: U.S. Department of Health, Education, and Welfare (1979b), Sec. 5, Life Tables.

early life are readily discernible for both white and non-white segments of the US population and for both sexes. Equally interesting are the relatively insignificant gains in life expectancy at ages over 40 or 50 (US Department of Health, Education, and Welfare 1979b; Section 5, Part A, pp. 5–13).

Impediments to longevity—smoking

It is my thesis that a significant retardant to improvement in life expectancy at the middle and later years of life is the entire category of environmental hazards in which I include certain personal habit patterns, particularly smoking, which hazards initiate or promote chronic processes exhibiting themselves in the middle and later years of life. This thesis is supported by a number of observations and findings in analyses of mortality data in relation to smoking.

In Table 2 it will be noted that the diseases related to tobacco use contributed 52.0 per cent of the total US mortality in 1975 (US Department of Health, Education, and Welfare 1979c; Section 1, Part A, pp. 1–100). Even if we consider only those entities for which a causal relationship is considered to be firm or highly probable, their contribution is still 41.0 per cent of total mortality.

In the Report of the Advisory Committee on Smoking and Health to the Surgeon General of the US Public Health Service, data derived from the Dorn (1958) study of US veterans could be utilized to compare the death rates by age among cigarette smokers and non-smokers. The results are presented in Fig. 1 (US Department of Health, Education, and Welfare 1964; p. 88). Throughout the age-scale cigarette smokers show a distinctly greater mortality than non-smokers and the ratios of smoker to non-smoker mortality are greater for the middle years of life. These data, however, do not take into account the varying

Table 2 Mortality from selected chronic diseases related to tobacco use: United States, 1975

Diseases	Number of deaths
Causally related:	
Cancer of lung, bronchus, trachea (162)†	82 040
Chronic bronchitis and emphysema (490–2)	23 507
Cancer of larynx (161)	3237
Cancer of lip (140)	158
Probably causally related:	
Coronary heart disease (410–13)	642 719
Cancer of bladder (188)	9369
Cancer of buccal cavity and pharynx (141–9)	7851
Cancer of esophagus (150)	6997
Possibly causally related:	
Cerebrovascular disease (430–8)	194 038
Aortic aneurysm (non-syphlitic) (441)	13 634
Total	983 550
Total mortality, all causes	1 892 879

†I.C.D. No.—International List of Causes of Death, Eighth Revision.
Source: US Department of Health, Education, and Welfare (1979c), Mortality Part A, Sec. 1.

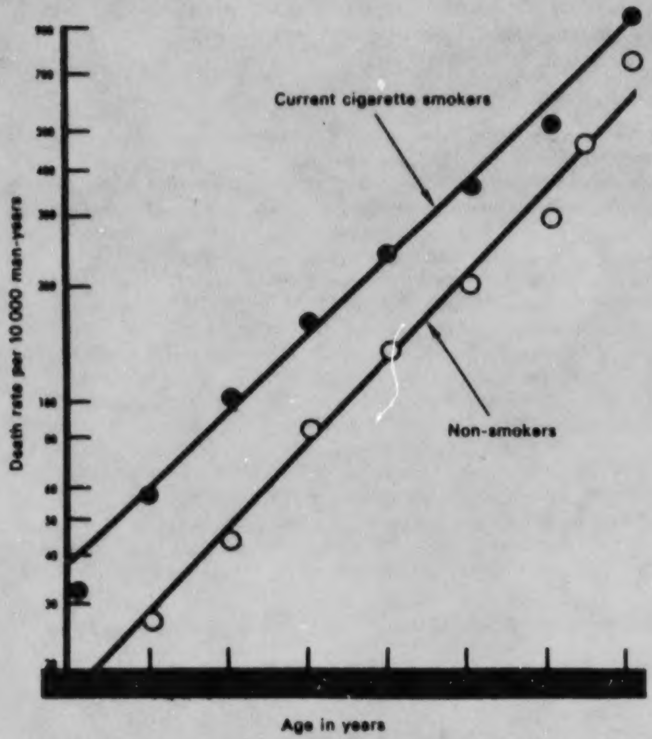

FIG. 1. Death rate (logarithmic scale) plotted against age—prospective study. of mortality in US veterans (US Department of Health, Education, and Welfare 1964).

contributions which smoking makes to disease-specific mortality, nor the percentage of smokers in the population. In some of these diseases the death rate differential (relative risk or mortality ratio) between smokers and non-smokers is far greater (e.g. lung cancer) than in others (e.g. coronary heart disease). Furthermore, even with a large death rate differential between smokers and non-smokers, a population with very few smokers would have very few excess deaths and a specific entity with a low overall death rate would likewise contribute very little excess mortality from the smokers affected by it. Thus, a combination of information is required to calculate the public health significance of smoking as a contributor to mortality in a given population. An indicator of the magnitude of the smoking problem would be the total excess deaths accounted for by smoking. These excess or additional deaths are those occurring per year among smokers above those deaths which would have occurred if smokers had had the same death rates as those who did not smoke. These additional deaths are expressed as a percentage of *all* deaths occurring in that age and sex group.

Horn (1967) utilizing the data derived from the Dorn study (Kahn 1966) and the 25-state study by Hammond (1966) calculated that for men between the ages of 35 and 60, approximately *one-third* of all their deaths would not have occurred if cigarette smokers had the same death rates as non-smokers. With the size of the smoking population in this age group, the impact of prevention of mortality on longevity by not smoking is obvious.

Cause-specific mortality

Table 3 presents the contributions which the several specific causes of mortality make to the excess deaths calculated as due to smoking in the seven large-scale prospective studies (US Department of Health, Education, and Welfare 1964; p. 108). It will be noted that although the relative risk for coronary artery disease among smokers is far lower than for lung cancer, the former contributes the largest number of deaths to the smoking excess. Lung cancer contributes the second largest amount followed by chronic bronchitis, emphysema, and other heart disease.

Early mortality

Early mortality of necessity reduces life expectancy for later years. Despite the popular misconception that smoking-related diseases produce mortality only at

Table 3 Percentage of total number of excess deaths of cigarette smokers due to different causes†

Underlying cause	British doctors	Men in 9 States	U.S. veterans	California occupational	California Legion	Canadian veterans	Men in 25 States
Coronary artery disease	32.9	51.9	38.6	43.5	43.5	44.2	51.7
Other heart disease	9.8	3.1	6.8	1.4	4.5	5.9	5.5
Cerebral vascular lesions	6.1	4.5	4.9	5.3	6.5	−1.8	3.3
Other circulatory diseases	1.9	2.7	7.1	1.7	0.2	5.6	4.4
Cancer of lung	24.0	13.5	14.9	20.2	16.8	18.3	13.6
Cancer of oral cavity, oesophagus, larynx	3.3	2.9	2.7	0.2	3.0	2.2	2.2
Other cancer	−0.2	9.8	8.9	6.3	−2.2	7.2	7.6
Bronchitis and emphysema	9.6	1.1	4.0	1.3	3.6	8.2	3.8
Influenza and pneumonia	−2.4	1.6	0.4	2.4	1.5	1.5	1.5
Stomach and duodenal ulcers	2.7	3.1	1.4	−1.7	2.2	2.9	1.3
Cirrhosis of liver	2.9	1.6	2.5	6.9	2.2	0.8	0.9
Accidents, suicides, violence	0.2	1.2	2.0	8.3	3.7	4.6	0.8
All other causes	9.2	3.0	5.8	4.2	12.5	0.4	3.4
All causes	100.0	100.0	100.0	100.0	100.0	100.0	100.0

†All cigarette smokers (current and ex-) for the two California and men in 25 States studies; current cigarette smokers only for the remainder.

Source: US Department of Health, Education, and Welfare (1964).

the extreme of life, the epidemiological evidence that smoking-related mortality is *premature* mortality is quite strong. Several lines of inquiry are available to us: One is the calculation by Hammond (1967), utilizing the data of the 25-state study of US males, of the loss in life expectancy among cigarette smokers as compared to the non-smokers in the study. A second is the analyses of excess mortality for several age groups of smokers by Horn (1967) as noted above.

Table 4 presents Hammond's data on the loss of life expectancy among those smoking different amounts of cigarettes per day. The data are in years lost as compared to the life expectancy of non-smokers at the several designated ages and also as a percentage of the total life expectancy of non-smokers. It can be noted that although the percentages of loss of life expectancy increase not only with quantity smoked per day but also with age, the absolute loss in years for any level of smoking is greatest among the younger age groups.

Table 5, modified from Horn's presentation, reveals that the proportions of excess mortality among both male and female smokers of cigarettes are highest

Table 4 Loss of life expectancy (in years and as a percentage of total life expectancy of non-smokers) at various ages for cigarette smokers, Hammond study US, 1967

	Number of cigarettes smoked per day							
Age	1–9		10–19		20–39		40 and over	
	Years lost	per cent	Years lost	per cent	Years lost	per cent	Years lost	per cent
25 years	4.6	9.5	5.5	11.3	6.2	12.8	8.3	17.1
30 years	4.6	10.5	5.5	12.5	6.1	13.9	8.1	18.5
35 years	4.5	11.5	5.4	13.8	6.0	15.3	7.9	20.2
40 years	4.3	12.5	5.2	15.1	5.8	16.8	7.6	22.0
45 years	4.1	13.7	5.0	16.7	5.6	18.7	7.0	23.3
50 years	3.8	14.8	4.6	18.0	5.1	19.9	6.3	24.6
55 years	3.5	16.4	4.0	18.7	4.4	20.6	5.4	25.2
60 years	3.1	17.6	3.5	19.9	3.9	22.2	4.4	25.0
65 years	2.8	19.9	2.9	20.6	3.1	22.0	3.4	24.1

Source: Hammond, E. C. (1967).

Table 5 Excess mortality among cigarette smokers as a percentage of all deaths in the respective age and sex groups. Dorn and Hammond studies (Kahn 1966; Hammond 1966)†

Study	Age				
	35–44	45–54	55–64	65–74	75–84
US veterans: men					
Excess deaths as per cent of total	33	43	21	17	8
Hammond: men					
Excess deaths as per cent of total	33	38	25	13	4
Hammond: women					
Excess deaths as per cent of total	5	9	4	2	—

†Modified from Horn (1967).

among the 45–54 year age group, next highest among the 35–44-year-olds, and then, in descending order of magnitude with increasing age from 55 years onward. For the females who also experienced the highest proportionate excess mortality related to smoking in the 45–54-year age group, the excess was of a lower magnitude, but significant nevertheless.

It was noted earlier that in an analysis of smoking mortality by specific cause of death the greatest contribution to the excess attributable to smoking was made by coronary heart disease. Although some of the large-scale prospective studies of mortality among smokers, such as the earlier Hammond and Horn (1958a, b) study, the Framingham studies by Doyle, Dawber, Kannel, Kinch, and Kahn (1964) and Kannel, Castelli, and McNamara (1968), and the Dorn US Veterans study reported by Kahn (1966), either did not have young enough subjects entering the studies or did not present analyses by age groups, a number of other large prospective studies provided data on coronary heart disease mortality for males and females in the age groups under 50. Notable among these are the Doll and Hill (1964) physicians study, the Best (1966) study in Canada, the 25-state study by Hammond and Garfinkel (1969), the Paffenbarger and Wing (1967) study, and the Weir and Dunn (1970) study in California. In virtually all of these the relative risk of coronary disease mortality for male smokers of cigarettes under the age of 50 and at the several levels of consumption was markedly higher than for the older age groups. In the large cohort of women in the later Hammond study (Hammond and Garfinkel 1969) a similar finding was noted. In a number of studies examining the role of smoking and its interaction with other risk factors for coronary heart disease in relatively younger men, cigarette smoking by itself was deemed a greater risk than the individual risks contributed by high serum cholesterol levels (Stamler *et al.* 1966), elevated systolic or diastolic blood-pressures (Borhani, Hechter, and Breslow 1963), obesity (Borhani *et al.* 1963), physical activity (Shapiro *et al.* 1969), and electrocardiographic abnormalities (Borhani *et al.* 1963). When smoking is combined with these other factors both additive and synergistic effects on mortality are noted. Thus mortality attributed to cigarette smoking, to which coronary heart disease makes the largest specific contribution, is distinctly a *premature* mortality which impacts itself on the prime years of life and in this period of life smoking is probably the greater risk factor in overall mortality.

Role of other factors

It cannot be denied that other factors influence disparities in mortality rates. It must be recognized that genetic or constitutional make-up plays a role. However, there are strong evidences that despite the influences of such variables the smoking factor exerts its own 'specific' strong effect on mortality. Until the 25-state study, few variables had been examined for this purpose and little information derived. The Hammond study provided data on such variables as longevity of parents and grandparents, religion, educational level, native or foreign birth, residence by size of town, occupational exposure, use of alcohol, use of fried food, use of tranquillizers, presence or absence of prior serious disease, marital status, and degree of exercise. Stratifying on each of these variables, age-adjusted death rates among those who smoked more than a pack of cigarettes a day and those who inhaled moderately or deeply were compared with those of non-smokers. In all instances, death rates were higher among individuals who smoked than those who did not. Several selected variables are presented in Table 6 (US Department of Health, Education, and Welfare 1964; pp. 100–1). Ipsen and Pfaelzer conducted further analyses of seven variables for the Surgeon General's Committee. None of these variables, with the exception of prior serious disease, had a stronger association with mortality than did smoking (US Department of Health, Education, and Welfare 1964; pp. 100–1). Hammond also conducted a special analysis for the Committee (US Department of Health, Education, and Welfare 1964; pp. 100–1) by matching pairs of cigarette smokers and non-smokers on the basis of height, religion, education, drinking habits, residence, and occupation. After 22 months of follow-up, mortality among the smokers was almost twice (1.86) that among non-smokers. Thus, the statement that smoking is a considerably stronger determinant of mortality than the variables tested, including those representative of constitutional differences, is warranted, particularly since adjustment for each of these variables individually

Table 6 Age-adjusted death rates per 1000 men (over approximately 22 months) for variables that may be related to mortality

Type of smoking	Long-lived parents and grandparents	Short-lived parents and grandparents	No previous serious disease	Previous serious disease	
None	14.8	21.1	11.5	42.5	
Cigarettes†	27.1	44.8	22.3	65.0	
	Single	Married	Use tranquillizers	Do not use tranquillizers	
None	26.0	18.9	29.1	18.2	
Cigarettes†	50.1	33.0	52.4	31.8	
	Educational level				
	No high school	Some high school	High school graduate	Some college	College graduate
None	22.7	20.0	16.9	18.3	15.8
Cigarettes†	35.2	34.5	35.5	34.2	20.4
	Degree of exercise‡				
	None	Slight	Moderate	Heavy	
None	23.8	14.7	11.0	9.5	
Cigarettes†	34.1	25.5	20.8	19.7	

†Smokers of more than a pack per day who inhaled moderately or deeply.

‡Confined to men with no history of heart disease, stroke, high blood-pressure or cancer (except skin) who were not sick at the time of entry.

Source: US Department of Health, Education, and Welfare (1964).

produced little, if any, change in the smoker–non-smoker mortality ratios. The implications for improvement of longevity are obvious.

Male vs. female mortality

Significant differences are observed in the overall mortality rates between males and females in the US population (US Department of Health, Education, and Welfare 1979c; Section 1, Part A, pp. 1–100). Such differences are particularly prominent for all of the smoking-related diseases discussed earlier. The disparities, with one exception, are in the direction of male excesses. In Table 7 it will be noted that the only exception to this is mortality ascribed to cerebrovascular disease. Otherwise the differences range from 5-fold for cancer of the larynx, 4-fold for both cancer of the lung and the lip, 3-fold for chronic bronchitis and emphysema, and for cancer of the oesophagus, 2.5-fold for cancers of the bladder and of the buccal cavity and for aortic aneurysm, to almost 1.5-fold for ischaemic heart disease.

Much speculation has attended these differences. To a certain extent sex hormonal differences in well-documented observations may account for a significant amount of the difference at ages prior to the menopause. This protective influence is noted in coronary artery disease, and is not specific since such sex disparities in susceptibility occur in poliomyelitis and hepatitis as well. In lung cancer the evidence is conjectural. Occupational exposures and similar differences in environmental exposure between the sexes may contribute to the disparities. Very often the disparities have been cited in attempts to discredit the basic association between smoking of tobacco and the relevant diseases. Such attempts have failed to take into account the disparities of tobacco exposure between males and females, which disparities have included not only intensity of smoking but history of initiation of smoking. Although some European populations, such

Table 7 Comparison of male and female cause specific mortality rates for selected chronic diseases related to tobacco smoking, US 1975

	Mortality rates/100 000	
Diseases	Male	Female
Causally related:		
Cancer of lung, bronchus, trachea (162)	61.1	17.0
Chronic bronchitis and emphysema (490–2)	17.5	4.9
Cancer of larynx (161)	2.6	0.5
Cancer of lip (140)	0.1	0.0
Probably causally related:		
Ischaemic heart disease (410–13)	348.8	257.0
Cancer of bladder (188)	6.4	2.5
Cancer of buccal cavity and pharynx (140–9)	5.5	2.2
Cancer of oesophagus (150)	5.0	1.6
Possibly causally related:		
Cerebrovascular disease (430–8)	81.3	100.4
Aortic aneurysm (non-syphlitic) (441)	9.4	3.5

Source: Department of Health, Education, and Welfare (1979c), Mortality Part A.

as the Finnish, were already smoking heavily in the 1880s, in the United States the main upsurge of cigarette consumption occurred approximately at the time of America's entry into the First World War. This increase was confined virtually entirely to the male population. The next major increase in cigarette consumption occurred during the Second World War when females began to participate extensively. Not only was the time of initiation of the astronomical rise in lung cancer in the male compatible with an induction period of 20-25 years following the First World War, but the acceleration of the rates in females was consistent with an induction period following their change in life-style in the Second World War and their adoption of the cigarette-smoking habit. A survey of smoking patterns by Haenszel in 1955 (Haenszel, Shimkin, and Miller 1956) noted that, at that time, twice as many males as females were smoking cigarettes, and males smoked considerably more cigarettes per day than females. The drift to younger ages for the initiation of the habit began earlier for males than females and inhalation practices were adopted later by the female. At the time of the survey the male to female ratio of lung cancer mortality was about 5:1. On correction for the disparities in the components of the smoking habits among males and females the ratio was reduced to 1.4:1. This residual may well be consistent with both hormonal protection and disparities in occupational and other environmental exposures between males and females. These data justify the conclusion that, at least for lung cancer and probably for other entities in which the relative risks among smokers are relatively large, sex disparities are predominantly the result of disparities in the smoking habits between the sexes.

Earlier in this paper it was noted that fully one-third of the mortality in our population of men between the ages of 35 and 60 would not have occurred if the non-smoker death rates had prevailed in this population (Horn 1967). The studies upon which these calculations were based were executed at a time when 57 per cent of the male and 28 per cent of the female population were current cigarette smokers. By 1966 smoking of cigarettes in persons 18 years and over had declined to 51 per cent in males and risen to 33 per cent in females (US Department of Health, Education, and Welfare 1970). In Table 8 (US Department of Health, Education, and Welfare 1970, 1971, 1976), the trend in cigarette smoking for males and females through the survey of 1975 can be noted. Male cessation of cigarette smoking has continued and is now true for all age groups over 18. This may, if it continues, be portentous for male survival and hence longevity. Declines in cigarette smoking in the younger age groups are especially noteworthy for those diseases with longer induction periods. For the older age groups, since cessation of smoking needs to have prevailed for 10 or more years to reduce mortality risk from coronary artery disease and lung cancer, little gain in survivorship can be expected here and now. However, improvement in survivorship among those with other entities more readily arrestable or reversible, such as the respiratory diseases associated with cigarette smoking, can be expected (Schuman 1971).

Table 8, however, reveals a gloomy picture for the female. In the 11-year period between 1955 and 1966 cigarette smoking prevalence actually increased

Table 8 Percentage of current smokers of cigarettes by sex and age. US surveys: 1955 and 1966 (Current Population Surveys-CPS) and 1970 and 1975 (Surveys conducted for National Clearinghouse for Smoking and Health-NCSH).

	Male				Female			
Age	CPS 1955	CPS 1966	NCSH 1970	NCSH 1975	CPS 1955	CPS 1966	NCSH 1970	NCSH 1975
18–24	53.0	48.3	47.0†	41.3‡	33.3	34.7	31.1†	34.0‡
25–34	63.6	58.9	46.8	43.9	39.2	43.2	40.3	35.4
35–44	62.1	57.0	48.6	47.1	35.4	41.1	39.0	36.4
45–54	58.0	53.1	43.1	41.1	25.7	37.3	36.0	32.8
55–64	45.8	46.2	37.4	33.7	13.4	23.0	24.3	25.9
65+	25.8	24.6	23.7	24.2	4.7	8.1	11.8	10.2

†Estimated.
‡21–4 years of age.
Sources: Haenszel *et al.* 1956; Department of Health, Education, and Welfare 1970, 1971.

among females in every age group and in one age group in particular, the 55–64-year-olds, by almost 50 per cent. Although declines in smoking prevalence occurred by 1970, and continued by 1975 in all but the oldest age-groups, the levels achieved did not equal those observed in 1955 except for those under 35 years of age. Increases actually continued to occur in the 55–64 age group. Any recidivism here may be expected to have a deleterious effect on female longevity.

Cessation of smoking and population mortality

The numerous prospective studies of general and cause-specific mortality and the case-control studies of specific smoking-associated diseases have left no doubt as to the benefits to be derived from cessation of cigarette smoking. In a review article, Schuman (1971) summarized these benefits. With specific respect to mortality, remarkable gains in survival were noted among ex-cigarette smokers both in terms of total mortality and by specific causes. Mortality ratios for ex-smokers declined, for example, an average of 63 per cent for lung cancer in 4 studies, 35 per cent for cerebrovascular disease, 33 per cent for coronary heart disease, and 28 per cent for chronic bronchitis and emphysema. In the instance of coronary heart disease this relatively modest gain compared to that of lung cancer is far more significant, since the *absolute excess* number of deaths from coronary heart disease attributable to smoking is far greater than that for lung cancer even though the proportion of lung cancers attributable to smoking is 90 per cent or more of the total load of such cancers.

These data do not take into account the interval since smoking was discontinued. For coronary heart disease, in men in the age group of 50–69 years, cessation of smoking of less than a pack a day yielded reductions in mortality in one to four years and, for a-pack-or-more-a-day smokers, in 5 to 9 years (Hammond 1966).

Public health benefits of cessation

Since, in the United States, declines in the proportion of male smokers over 18 years of age have occurred over the period 1955–75, certain impacts on mortality might be detectable. Several complicating factors must be considered in assessing any changes, however. Since *per capita* consumption of cigarettes declined from 1966 to 1970 following the release of the Report of the Advisory Committee to the Surgeon General and since women, particularly young women, had increased their consumption, the decline signified a marked decrease in consumption by men.

It is of interest that two categories of disease with relatively rapid 'turn-around' properties in relation to tobacco smoking declined significantly. Whereas coronary heart disease in men had been increasing over the previous two decades, in 1966 there began a reversal of this trend which continues. No such decline has been noted in women. It would be tempting to ascribe this reversal to the reduction in smoking, but, as the relative risk for smokers is of far smaller magnitude in coronary heart disease than in lung cancer, the evidence is not clear-cut and it is possible that other causal or risk factors not currently being surveyed in the population may also be declining. However, it would be somewhat difficult to assume a change in the latter factors operating solely in males. The natural experiment invoked by the cessation of smoking among British physicians (Fletcher and Horn 1970) yielded a 6 per cent reduction in total cardiovascular mortality in an eight-year period.†

Similarly male death rates from chronic bronchitis and emphysema have been declining since 1967, whereas female death rates have not declined.

Lung cancer with its high relative risk among smokers would be a sensitively responding disease since more than 90 per cent of all such cancers are attributable to cigarette smoking. However, we are faced with several complicating factors. The induction period being relatively long, response to a decline in smoking would lag significantly. Furthermore, the declines in consumption have been proportionately greater in the younger age groups in which switches to filter cigarettes and those with lower tar and nicotine have also occurred. In these age groups the lung cancer rates are normally low. Further data will be necessary over the next several years for an appraisal of the groups of males born after 1919, which was the last birth cohort to reach the peak of cumulative cigarette exposure. Cumulative exposure for birth cohorts since then has been declining. A suggestive decline in lung cancer among these younger males has already been noted.

Longevity and quality of life

I turn now to what I deem to be the more significant aspect of longevity. The ultimate aim of this Conference on Aging has most appropriately been expressed as the improvement of the quality of life for older people in society. Certainly longevity without productivity, sustained interest, reciprocal appreciation of the life about us, without a contribution to humanity and the joy of living is not life. The saving of life alone is not enough. The prolongation of life without quality is a questionable goal. Thus, life for those whose demise has been postponed, but who suffer the ailments and disabilities induced by smoking is certainly of inadequate quality.

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† In a more recent paper published by Doll and Peto, after this chapter was written, a 25 per cent reduction in ischaemic heart disease mortality was noted for smokers aged 30–54 years discontinuing for more than 15 years.

A large number of case-control and cohort studies on morbidity prevalence and incidence in relation to smoking can be found in the literature. As with mortality studies the association of tobacco smoking with a number of cardiorespiratory entities representing serious and disabling states has been well documented and the declines in *morbidity* ratios upon cessation of smoking summarized (Schuman 1971). Parallel with total mortality excesses among smokers, an over-all measure of morbidity is excess disability among smokers as measured by days lost from work, days of restricted activity, and days confined to bed. Information on excess morbidity related to smoking has become available through periodic inquiries on smoking among those in probability samples of the ongoing National Health Survey (Department of Health, Education, and Welfare 1967). This source indicates that for all three types of disability measures noted above and for both men and women, higher morbidity rates, higher morbidity ratios, and higher percentages of excess disability days were recorded for cigarette smokers.

In Table 9, a modification of the data as calculated by Horn (1967), it will again be noted that, as for mortality, the excess disability days among cigarette smokers were found to be proportionately greater in the younger age groups. For males the greatest excess in each of the three disability measures is in the 45–64-year age group. For females it is in an even younger group—17–44 years of age. The same data source provided information on prevalence of chronic conditions. Among smoking men and women, the youngest age groups (17–44 years) showed the highest proportion of excess prevalence. For all these measurements a dose-effect gradient with the number of cigarettes smoked per day was noted. Thus smoking is also related to *prematurely* disabling illness.

Table 9 Excess morbidity among cigarette smokers as a percentage of all disability days in the respective age and sex groups. National Health Survey (US Department of Health, Education, and Welfare 1967)†

	Males			Females		
Disability measure	17–44	45–64	65+	17–44	45–64	65+
Work-loss days	20	28	0‡	18	11	§
Restricted activity days	23	28	8	14	5	2
Bed days	23	28	−1	10	6	0

†Modified from Horn (1967).

‡0 indicates no difference in rates between smokers and non-smokers.

§Too few smokers.

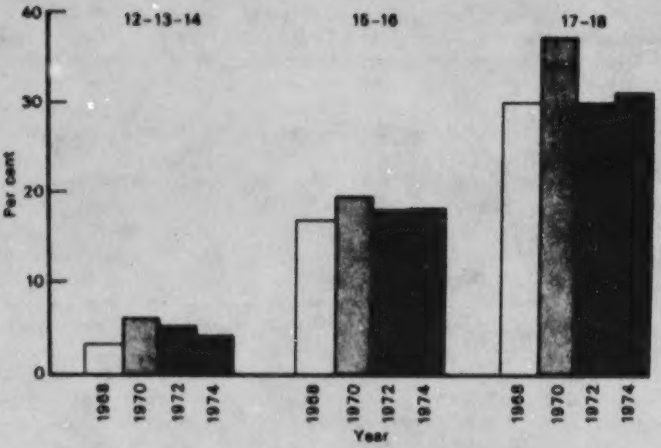

FIG. 2. Per cent current regular smokers—teenage boys, 1968–74.

The literature is also replete with evidence of the reversibility of the pathology of early bronchopulmonary entities (Huhti 1965; Coates, Bower, and Reinstein 1965; Holland 1966; Higgins, Gilson, Ferres, *et al.* 1968; Holland and Elliott 1968; Fletcher 1968; Comstock *et al.* 1970; Wilhelmsen 1967; Peterson *et al.* 1968; Auerbach *et al.* 1962, 1963). Thus, not only will abstinence from smoking prevent both early morbidity and mortality, but cessation of smoking will materially reduce the risks of development of the specific smoking-related diseases in those now smoking, thus increasing longevity and reversing the process in some diseases with elimination of disabling illness.

Unfortunately a note of pessimism must be interjected if only to evoke attention to a social imperative. Very recent surveys on patterns of cigarette smoking in the US population from ages 12 to 18 reveal the disturbing fact that although the percentage of current regular smokers among boys aged 12 to 14 has declined somewhat from 1970 to 1974, the percentages have plateaued at relatively high levels for boys aged 15 to 16 and 17 to 18 (Fig. 2) and for girls at all ages the percentages of smokers have steadily increased in every year between 1968 and 1974 (Fig. 3).† If these are the cohorts of the future, then the risk of thwarting improvements in longevity is great.

To paraphrase a conclusion from the Report on Smoking and Health by the Advisory Committee to the Surgeon General which is just as timely today: 'Cigarette smoking continues to be a health hazard of sufficient importance to warrant appropriate immediate remedial action.'

† Teen-age smoking—national patterns of cigarette smoking, ages 12 through 18, in 1972 and 1974. DHEW Publication No. 76-931.

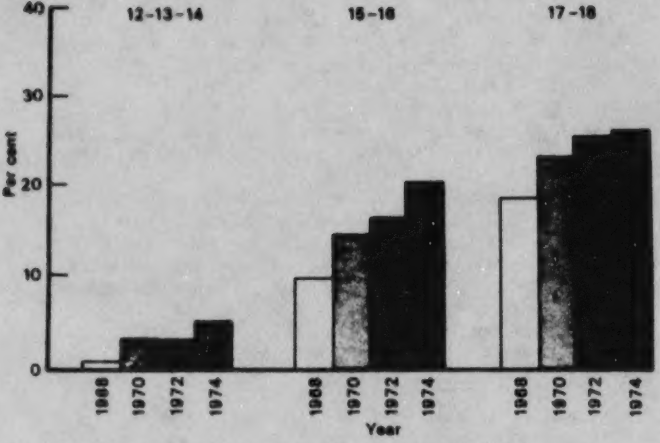

Fig. 3. Per cent current regular current smokers—teenage girls, 1968–74.

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Discussion—Session 12 Alexander Leaf

An anonymous prescription for a long life states, 'Choose your parents carefully!' The importance of genetic factors in determining the life span becomes apparent when differences in longevity among species are compared. Rats very rarely exceed four years; cats, thirty years; horses, forty years; elephants, sixty years.

The Benefits of Cessation of Smoking

*Leonard M. Schuman, M.D.**

The overwhelming epidemiologic, clinical and laboratory evidences for the relationship of cigarette smoking to disease have been so extensively documented and evaluated[1-6] and so widely disseminated through the world scientific literature and the press that it would be superfluous to review them extensively at this time. However, a brief display of selected basic observations will assist in orienting our approaches to the problems of the smoking patient.

The magnitude of the problem, demonstrable in many countries of the world and demanding increased attention from practitioners of medicine and serious response from the population, is exemplified by data from the United States. The diseases associated with tobacco use are presented in Table 1. These diseases contributed 48 percent of the total U.S. mortality in *1967*. If we restrict ourselves solely to those diseases for which a causal relationship with tobacco use is deemed to be firm or highly probable, they contributed *37* percent to total mortality. These, however, are inclusive data: they do *not* separate the smoking segment of the population nor the contribution which smoking makes to disease–specific mortality. We need an indicator of *public health significance*—a measure of the number of people affected by the factor and hence a measure of the magnitude of the problem for the total population. Such an indicator is the excess deaths among smokers over those among nonsmokers as a percentage of total deaths in the group. This measure takes into account not only the differences in death rates between smokers and nonsmokers but also the proportion of smokers in the population under study. This is important to the consideration of public health significance for it is obvious that even with a large death rate differential between smokers and non-smokers a population with very few smokers would have very few excess deaths.

Utilizing the data from the prospective studies on smoking among veterans by Dorn and the 25-state study by Hammond, Horn determined[4] that, for men between the ages of 35 and 60, approximately *one-third* of all their deaths would not have occurred if cigarette smokers had the same death rates as nonsmokers.[4] The potential for prevention is immediately obvious.

To the smokers among the very young such data frequently leave them unresponsive for in their minds the mortality producing diseases related to smoking are diseases of the extreme end of life—so far in the future as to pose no threat at all or, at worst, a small price to pay for the pleasures of today. To the inveterate smokers among the population groups in the prime years of life's productivity—ages 45-49—such data may well evoke the response: ". . . but we all have to die sometime and from something." To such smokers the physicians' armamentarium need not be without response, for he has at his command quite solid epidemiologic evidence that mortality related to smoking is *premature* mortality. The evidence is available in two

Table 1—***Mortality from Selected Chronic Diseases Related to Tobacco Smoking, United States, 1967***

Diseases	Number deaths
Causally related:	
Cancer of lung, bronchus, trachea (162,163)	54,407
Chronic bronchitis and emphysema (501,502,527.1)	25,181
Cancer of larynx (161)	2,797
Cancer of lip (140)	143
Probably causally related:	
Coronary heart disease (420)	573,153
Cancer of bladder (181.0)	8,563
Cancer of buccal cavity and pharynx (141-148)	6,575
Cancer of esophagus (150)	5,627
Possibly causally related:	
Cerebrovascular disease (330-334)	203,164
Aortic aneurysm (451)	11,621
Total	891,251
Total mortality, all causes	1,851,323

Source: Vital Statistics of the U.S., 1967, vol. II, Mortality, Part A.[6]

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*Professor and Head, Division of Epidemiology, School of Public Health, University of Minnesota, Minneapolis, Minn.

Table 2—Loss in Life Expectancy (in years and as a percentage of total life expectancy of non-smokers) at Various Ages for Cigarette Smokers Hammond Study U.S., 1967[7]

Age	Number of Cigarettes Smoked per Day							
	1-9		10-19		20-39		40 and over	
	Years Lost	%	Years Lost	%	Years Lost	%	Years Lost	%
25 years	4.6	9.5	5.5	11.3	6.2	12.8	8.3	17.1
30 years	4.6	10.5	5.5	12.5	6.1	13.9	8.1	18.5
35 years	4.5	11.5	5.4	13.8	6.0	15.3	7.9	20.2
40 years	4.3	12.5	5.2	15.1	5.8	16.8	7.6	22.0
45 years	4.1	13.7	5.0	16.7	5.6	18.7	7.0	23.3
50 years	3.8	14.8	4.6	18.0	5.1	19.9	6.3	24.6
55 years	3.5	16.4	4.0	18.7	4.4	20.6	5.4	25.2
60 years	3.1	17.6	3.5	19.9	3.9	22.2	4.4	25.0
65 years	2.8	19.9	2.9	20.6	3.1	22.0	3.4	24.1

forms: loss in life expectancy and excess mortality in the several pertinent age groups.

Hammond, from the data on the U.S. men in his 25-state study, utilizing the life expectancy of the men who never smoked regularly as the standard, was able to calculate the loss in life expectancy at various ages among the regular cigarette smokers. Table 2 presents these data.[7] In any single age group the *percentage* of life expectancy lost among smokers increases with the amount of cigarettes smoked per day, and at any level of smoking the *percentage* of life expectancy lost increases with age. However, from these data it can readily be seen that the greater absolute number of years lost in life expectancy occurs among the younger age groups for any level of smoking.

Both the Dorn[8] study of veterans and the Hammond[9] study of over 1,000,000 men and women in 25 states provided data for Horn's calculations of excess deaths among smokers as percentages of total mortality in the relevant age and sex groups.[3] Excess deaths among smokers were the number of deaths over and above those which would have occurred if smokers had the same death rates as those who never smoked regularly. Although there is an excess mortality among cigarette smokers in each age group, Horn's calculations reveal these smoking-related excesses to be proportionately higher for the younger age groups. In both studies the male excess smoker mortality was proportionately greatest in the 45-54 year age group. This excess was 43 and 38 percent respectively of the total mortality in this age group. The second highest proportionate excess was in the 35-44 year age group (33 percent in both studies.) In the age groups beyond 54 years the proportionate excesses declined progressively.

Women smokers in the Hammond study also experienced their highest proportionate excess mortality in the 45-54 year age group. This excess was of a lower magnitude than for the men, but was still a significant 9 percent of their total age-specific mortality. Thus, the impact of smoking-related mortality is felt predominantly in the prime years of life for both men and women. This is evidence enough to appeal to man's evocable, albeit subconscious, striving for a measure of immortality.

We must, in turn, not overlook a possibly more immediate patient concern and that is illness which disables for longer or shorter periods, which reduces his productivity and restricts even his more or less spiritual activities—in short, which reduces the quality of life for him and his family. Information on excess morbidity related to smoking has become available through periodic inquiries on smoking among those in the probability samples of the ongoing National Health Survey.[10] In this survey, disabling illness has been measured in three ways: days lost from work, days in which activity has been restricted, and days confined to bed. For all three types of disability and for both men and women, higher morbidity ratios, greater morbidity rates and higher percentages of excess disability days are found among cigarette smokers.[5] As with mortality, excess disability days among smokers of cigarettes is greater, proportionately, in the relatively younger age groups. Among men smokers, the 45-64 year age group reported the highest excess morbidity—a 28 percent excess of disabling illness by each of the three morbidity measures. The 17-44 year age group was next with a 20-23 percent excess. Among women smokers, disabling illness occurred in highest excess in an even younger age group—those 17-44 years of age—with the age group 45-64 showing the next highest morbidity excess.

The National Health Survey also provides data on prevalence of chronic conditions among smokers and non-smokers. It is of further interest not only that the smokers have higher prevalence rates of chronic conditions, but that the excess prevalence of such chronic conditions among young smokers (17-44 years of age) is proportionately higher than for all older age groups.[10] This is true for both men and women. For both the three measures of disability and the prevalence rates of chronic conditions a dose-effect gradient with amount of cigarettes smoked per day was demonstrated. It is thus quite apparent that smoking is also strongly related to *prematurely* disabling illness.

Despite these evidences of the gravity and magnitude of the problem the physician will frequently share the pessimism of the long-time smoker, the heavy smoker who questions the value of *cessation* of smoking—who himself states "I've been smoking so long, the damage has been done. What can be gained by stopping?" Data from the prospective studies would tend to refute this pessimism for these reveal repeated evidences of advantageously lower risks of mortality among smokers who have discontinued the habit than among continuing smokers. In the first portion of Table 3, general mortality among smokers and ex-smokers of cigarettes is expressed as a ratio relative to mortality in non-smokers. For each of the five studies in which the classes of smokers could be separated, ex-cigarette smokers had distinctly lower risks than continuing smokers.

Of importance to the skeptical patients who believe that their situation is hopeless for having smoked excessively for a great number of years is the reduction in mortality risk noted in several studies after cessation of smoking.[2] Even if the patient has smoked more than a pack a day for 25-35 years, his risk of death is materially reduced. Even if he has smoked more than a pack a day, very significant gains are apparent if he quits at ages 45-54. If he smokes no more than a pack a day then, even at ages over 55, significant reduction in risk is evident. If he remains a discontinued smoker for five to ten years, his risk of death is reduced almost to that of one who has never smoked.[11]

Gains in reduction of disease-specific mortality by cessation are even more dramatic for several of the specific entities associated with cigarette smoking. Even with the relatively short term followup data available during the evaluation of the prospective studies for the Advisory Committee's Report to the Surgeon General,[2] significant reductions in risk on cessation of smoking had been noted for all but one of the several diseases associated with cigarette smoking. Chronic bronchitis and emphysema mortality was actually higher among ex-smokers than current cigarette smokers. (This was a phenomenon also noted in the total mortality rates for discontinued pipe and cigar smokers at that time.)

Since the data on ex-cigarette smokers were generally not adjusted for duration of discontinuance or for reasons for discontinuance (eg doctor's orders or severity of already-existing illness) this apparent discrepancy in an opposite direction was probably an artifact. Actually, it can be shown by more recent data derived from longer term follow-up in at least two studies[8,14] that reduction in risk of mortality from chronic bronchitis and emphysema does indeed occur with cessation of smoking cigarettes.

Table 3 presents these recent data by specific prospective study and for specific disease entities. A remarkable consistency in the reduction of mortal-

Table 3—Comparison of Mortality Ratios of Ex-Cigarette Smokers with Current Cigarette Smokers

Entity	Mortality Ratios	Current Cigarette Smokers	Ex-Cigarette Smokers
Total Mortality (2)			
British physicians (Doll and Hill)		1.44	1.04
Men in 9 States (Hammond and Horn)		1.70	1.40
U.S. veterans (Dorn)		1.79	1.41
Canadian veterans (Best et al)		1.65	1.42
Men in 25 states (Hammond)		1.83	1.50
Cause—Specific Mortality			
Lung Cancer			
U.S. veterans (Dorn) (8)		12.1	5.0
25 states (Hammond) (9)	1-19cigs/day	6.5	2.0
	20 +	13.7	7.9
Canadian veterans (Best et al) (13)	1-9	10.0	6.1
	10-20	16.4	
	21 +	17.3	
British physicians (Doll and Hill) (14)		17.4	3.4
Chronic Bronchitis and Emphysema			
U.S. veterans (8)		10.1	7.8
British physicians (14)		11.6	7.8
Cancer of Larynx (2)		7.1	5.4
Cancer of Oral Cavity			
U.S. veterans (8)		4.1	2.1
Cancer of Esophagus			
U.S. veterans (8)		6.2	1.6
Cancer of Bladder			
U.S. veterans (8)		2.2	1.6
Coronary Heart Disease			
U.S. veterans (8)		1.7	1.3
25 states (11)	1-19cigs/day	1.9	1.2
	20 +	2.6	1.5
British physicians (14)		1.4	1.1
Cerebrovascular Disease			
U.S. veterans (8)		1.5	1.3
25 states (11)	1-19cigs/day	1.5	1.0
	20 +	1.9	0.9
Aortic Aneurysm (non-syphilitic)			
U.S. veterans (8)		5.2	2.0
Cirrhosis of the Liver			
U.S. veterans (8)		3.3	1.1

ity ratios among ex-cigarette smokers for each specific disease is apparent among the several studies. The extension of the studies over a longer period of followup minimized the impact of the earlier higher mortality among those who discontinued smoking because of severe illness. In the U.S. veterans study, with the largest number of accumulated deaths (over 46,000) of any of the prospective studies, the volume of the data and the foresight of the investigator permitted the development of a subcategory of discontinued smokers who stopped smoking for reasons other than "doctor's orders." The data on ex-smokers of cigarettes derived from the U.S. veterans study are for this category of discontinued smokers.[8]

In the most recent analyses of the Hammond 25-state data, the investigators confined themselves to the study of the approximately 80 percent of their subjects who, at the time of enrollment, had no history of heart disease or stroke, no history of cancer diagnosed within the preceding five years and were not sick.[11] In this manner smokers who had discontinued smoking because of ill health prior to the time of enrollment were excluded.

It is noteworthy that the greatest reductions in risk are to be found for the cancers associated with cigarette smoking. This is not surprising when it has been noted that the greater the magnitude of the relative risk the more likely that cigarette smoking is the principal causal factor in the disease. Lower relative risks imply other related factors may also be causally operating.

Because of the relatively small mortality ratios observed for coronary heart disease, the magnitude of the gains from cessation of smoking cigarettes may be inapparent on superficial inspection. Cornfield and Mitchell[12] hold a conservative attitude with regard to the degree of decrease in risk with smoking discontinuance. They cite the persistence of some coronary mortality effect after at least a decade of discontinuance of smoking, but do acknowledge significant reductions in risk. Because of the magnitude of the coronary heart disease problem, what appears as a small reduction in ratio is a highly significant reduction in absolute death rates from this cause. Table 4 demonstrates this point. The differences between the rates for current smokers and for ex-smokers represent the gains for the specific levels of smoke exposure and for the several age groups. As an illustration, in the 55-64 year age group, discontinued smokers who had smoked more than two packs of cigarettes per day achieved a coronary heart disease mortality risk level which was 455 deaths per 100,000 *less* than those who continued to smoke at that dose-rate. This latter smoking group experienced a coronary heart disease mortality *rate* of 1101 per 100,000. Thus, a 41 percent reduction in mortality risk is certainly meaningful.

This can also be seen in the 25-state study of Hammond with respect to the length of time smoking of cigarettes had been discontinued and the level of smoking which had prevailed prior to such discontinuance. In Table 5 it can be seen that, for both levels of smoking, the longer the discontinuance, the greater the reduction in risk, and after ten years the risk is, respectively, equal to or almost equal to that of those who never smoked.

Since there continue to be those who would refute these data with the charge of selective bias, nature has obligingly produced a situation similar to a controlled cessation experiment. In England and Wales, a considerably large reduction in cigarette smoking among physicians followed upon the first Doll and Hill report in 1954, whereas among the general population, cessation of smoking was not a common phenomenon. In the general population, increases in mortality from lung cancer and cardiovascular diseases have taken place in the 10- to 11-year period following this report. Whereas the lung cancer mortality in the general male population, aged 35-84, increased from 1.49 to 1.86 per 1000 population per year between the periods 1954-57 and 1962-64, an increase of 25 percent, the lung cancer mortality among the same-aged British physicians actually declined from 1.09 to 0.76 per 1000

Table 4—Annual Death Rate per 100,000 from Coronary Heart Disease by Age, Cigarette-Smoking Status and Number of Cigarettes Smoked per Day, U.S. Veterans Study (8)

Number smoked per day[1]	45-54 Current cigarette smokers	45-54 Ex-smokers[2]	55-64 Current cigarette smokers	55-64 Ex-smokers[2]	65-74 Current cigarette smokers	65-74 Ex-smokers[2]
1 to 9	195	128	594	432	1,374	1,108
10 to 20	297	133	830	557	1,577	1,200
21 to 39	390	57	912	743	1,701	1,266
40 +	502	—	1,101	646	1,965	1,482

[1] This is the current rate of smoking for current cigarette smokers and the maximum rate attained for ex-cigarette smokers.
[2] Ex-smokers who stopped for reasons other than doctor's orders.

Table 5—Coronary Heart Disease (Men). Age-Standardized Death Rates for Ex-Cigarette Smokers with History of Cigarette Smoking Only, by Former Number Smoked per Day and Years Since Last Cigarette Smoking. Death Rates for Current Cigarette Smokers with History of Cigarette Smoking Only and Men Who Never Smoked Regularly are Shown for Comparison. Men Aged 50-69[7]

Ex-cigarette smokers (years since last cigarette smoking)	Smoked 1-19 cigarettes a day: Number men	Smoked 1-19 cigarettes a day: Number deaths	Smoked 1-19 cigarettes a day: Death rate	Smoked 20+ cigarettes a day: Number men	Smoked 20+ cigarettes a day: Number deaths	Smoked 20+ cigarettes a day: Death rate
Under 1 year	746	27	[1]1,006	2,244	77	[1]1,070
1 to 4 years	1,844	51	718	5,435	195	1,003
5 to 9 years	1,770	48	725	5,803	152	732
10+ years	4,209	84	498	8,147	206	679
Total ex-smokers	8,569	210	635	21,624	630	813
Current cigarette smokers	22,908	781	947	56,585	1,895	1,029
Never smoked regularly	55,728	1,114	502	55,728	1,114	502

[1] Four or more but less than 10 deaths expected in some of the component 5-year age groups.

population in the same period; a *reduction* of 30 percent.[13] Similarly, whereas the total cardiovascular disease mortality among the general male population aged 35-64 increased 10 percent between the periods 1953-57 and 1961-65, mortality from these diseases actually declined by 6 percent among the British physicians.[14]

May I once more turn your attention from mortality to illness studies, for it is in this area of maintenance of health and alleviation of symptoms of disease that a more successful appeal to the smoker may probably be made. A large number of prospective studies in the United States and abroad have directed their attention to *coronary heart disease* incidence and morbidity. Notable among these in the United States are the studies at Framingham, Massachusetts and Albany, New York; the Peoples Gas and Light Co. and Western Electric Co. studies in Chicago, Illinois; the five-county study in North Dakota; the Western Collaborative Study; the Health Insurance Plan Study in New York City; the Los Angeles Heart Study; and the Tecumseh, Michigan, Study. The prospective and retrospective studies of Dorken, Friedmann, and Schimmler, respectively, in Germany, Mulcahy in Ireland, Medalie in Israel, Hyams in Japan, Natvig in Norway and Heyden-Stucki in Switzerland are also of significant note.

From among these studies several have gathered sufficient data permitting analyses of incidence rates for current and discontinued smokers. Table 6 reveals the decline in risk with discontinuance of cigarette smoking in three prospective studies. In the Western Collaborative Group Study this decline was significant for the 50-59 year age group only. In the younger age group the risk among discontinued smokers was approximately the same as for the continuing moderate to heavy smokers. However, since no information is available on reasons for discontinuance nor length of time of such discontinuance, the meaning of this lack of difference in the younger age group cannot be determined.

Morbidity from *bronchopulmonary entities* is probably most evident to the smoker even if he is suffering from relatively mild pathology such as a chronic productive cough. Furthermore, it is with this group of disease entities that remarkable diminution or disappearance of symptoms follow rapidly after cessation of smoking.

We are indebted to the National Health Survey for data on the prevalence of chronic bronchitis and/or emphysema among smokers of cigarettes, non-smokers and ex-smokers.[19] Among young men aged 17 years and over, ex-smokers revealed an age-adjusted prevalence rate of 2.5 cases per 100 as compared with 3.3 cases for current smokers of

Table 6—Comparison of Risks of Developing Coronary Heart Disease Among Continuing and Ex-Smokers of Cigarettes

Study (Reference)	Relative Risk (non-smoker = 100): Current Cigarette Smokers	Relative Risk (non-smoker = 100): Ex-Cigarette Smokers
Framingham and Albany[17]*		
<20 cigs/day	179	107
20 cigs/day	185	
>20 cigs/day	274	
North Dakota [18]*	231	107
Western Collaborative Group [16]**		
a) 39-49 year age group:		
1-15 cigs/day	142	266
16-25	247	
26+	373	
b) 50-59 year age group:		
1-15 cigs/day	134	104
16-25	186	
26+	238	

*Myocardial infarction only.
**All coronary heart disease. Calculated from authors' data

more than a pack per day. Among women, the age-adjusted prevalence for ex-smokers was 2.6 cases per 100 as compared with rates of 4.0 per 100 for smokers of one-half to one pack a day and 6.5 per 100 for smokers of more than a pack a day—an even more favorable reduction in risk. These data of the National Health Survey are of special interest, since it is entirely likely that the real effects of cessation of smoking may in part be masked by the possibly greater tendency for discontinuance among those disabled by severe bronchopulmonary disease.

When one turns to symptoms and pulmonary function tests rather than finite diagnoses with their difficulties of definition, the benefits of cessation are more readily discernible. A number of surveys have demonstrated much lower proportions of individuals with chronic cough and with phlegm among ex-smokers than among those continuing cigarette smoking—proportions which approach the prevalence in non-smokers. One such study of a total regional population in Finland by Huhti[20] provides the data for Table 7 which is illustrative of many. Markedly lower prevalences of cough and phlegm among men ex-smokers are noted even in comparison with the continuing smokers of small numbers of cigarettes per day. Similar contrasts are noted for women ex-smokers, but the continuing smokers of 15 or more cigarettes per day among women were too few for a statement of significance.

Coates et al,[21] in their study of Detroit postal workers, found the ex-smokers to have prevalences of chronic cough and phlegm equal to that of non-smokers. Holland,[22] in a study of van-drivers in London and rural towns in England and in East Coast towns of the United States, found the proportions with chronic cough and phlegm among ex-smokers of cigarettes much closer to that in non-smokers than among even the continuing *light* smokers (from 1-14 cigarettes per day) of cigarettes.

Table 7—Percent of Men and Women with Cough (3 months in the year) and with Phlegm (3 months in the year) Related to Smoking Habits (20)

	Cigarettes smoked per day			Non-smokers	Ex-smokers
	1-14	15-24	25 +		
Cough					
Men	31.5	40.8	42.1	4.1	8.5
Women	10.4	3 of 7 women smoking 15 + cigarettes/day		4.5	13.3
Phlegm					
Men	38.0	42.9	42.4	10.7	17.7
Women	10.4	4 of 7 women smoking 15 + cigarettes/day		5.9	13.3

In some of these prevalence studies pulmonary function tests accompanied the medical questionnaires. Here also the ex-smoker of cigarettes showed values for 1-second forced expiratory volume and peak expiratory flow rates significantly higher than those for continuing smokers and almost as high as the values for non-smokers.[20, 23]

Evidences from these cross-sectional studies suggest the inference that cessation of cigarette smoking in the individual brings improvement in pulmonary function and diminution or eradication of chronic respiratory symptoms. Support for such an inference is found in longitudinal studies which re-examined the same subjects at a reasonable interval of time. The studies by Higgins and associates,[23] Holland and Elliott,[24] and Fletcher[25] are examples of note. More recently Comstock and his co-workers,[26] in a study of men telephone company employees re-examined after a five-year interval, found marked improvement in the prevalence of cough and phlegm among those who had quit smoking cigarettes between the two surveys. Furthermore, though $FEV_{1.0}$ values for all smoking classes declined between examination rounds, the ex-smokers of cigarettes showed the smallest decline and thus more closely approached the value for the non-smokers.

Experimental evidence also supports these findings. The studies of Krumholz et al,[27] with smoking subjects who abstained for six weeks, revealed significant increases in peak flow rates, diffusing capacity, inspiratory reserve volume and maximal voluntary ventilation. Heart rate, oxygen debt after exercise and functional residual capacity was decreased. Wilhelmsen[28] demonstrated marked decreases in coughing, sputum production and wheezing in a group of long term smokers who abstained from cigarette smoking for 40 days. A significant increase in $FEV_{1.0}$ also occurred. Peterson and co-workers[29] similarly demonstrated significant increases in pulmonary function and decreases in coughing and breathlessness among smokers of cigarettes after 18 months of abstinence.

Finally, autopsy materials on discontinued smokers reviewed by Auerbach et al[30] reveal changes in the tracheobronchial tree (such as loss of cilia, basal cell hyperplasia and atypical cells) in quantitative proportions more nearly like that of non-smokers as opposed to moderate or heavy smoker patterns. Similarly, these same investigators found a lesser degree of pulmonary fibrosis, rupture of alveolar septa and thickening of the walls of small arteries and arterioles in the pulmonary parenchyma of individuals who had stopped smoking cigarettes for five years or more than among current smokers.[31]

Thus, there is good evidence of reversal of bronchitic changes and some intimation of reversal of parenchymal change or at least arrest.

In summary, it can be said with great optimism that cessation of smoking does indeed reverse a number of processes and arrest others, short of morbidity or, at worst, premature mortality. To the benefits in terms of reduction of disease and disability must be added the increase in comfort, the feeling of well-being and positive health, not to mention the esthetic gains of a fresh smelling breath and a clear atmosphere. This optimism can and must be carried into office practice and communicated as prognosis to the scoffing young smoker and the inveterate older chronic smoker.

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Reprint requests: Dr. Schuman, 1158 Mayo Memorial Building, University of Minnesota, Minneapolis 55455.

Approaches to Primary Prevention of Disease

LEONARD M. SCHUMAN, M.D.

Representatives of the U.S. Congress, the Public Health Service, schools of public health, schools of medicine, labor, and industry met on February 27, 1968, to discuss the need for study of the total problem of disease prevention. The outcome of this meeting was the establishment of the Advisory Committee on Health Protection and Disease Prevention to the Secretary of Health, Education, and Welfare. The paper presented here is based on a talk which Dr. Schuman gave at the meeting and which served as the base of a position paper presented to President Lyndon B. Johnson.

MAN INSTINCTIVELY mends cracked foundations, repairs broken fences, plugs holes in leaky roofs, binds cracked and drooping tree limbs, and, in turn, palliates pain, dialyzes blood for malfunctioning kidneys, and replaces heart valves or even hearts ravaged by disease. Equally instinctively man flinches from a threatened blow, runs from a burning building, turns from a dangerous precipice, and shies from persons with the defacing lesions of an infectious disease. Each act is one of preservation. Each is directed toward avoidance of an uncomfortable and even disastrous outcome. Each is prevention, yet with distinct and grossly understressed differences in goals and efficiency of attainment.

In the health field, the excision of a tumorous lung is an attempt to cure or arrest the disease or postpone death, and physical therapy and physical rehabilitative procedures for the stroke victim are attempts to minimize disability. The erection of barriers between radiative sources and man is designed to obviate the occurrence of radiation sickness and leukemia, and administration of a specific vaccine will obviate the occurrence of poliomyelitis. Although each approach shares the common characteristics of man's striving for a measure of immortality, there are obvious, distinct differences in the philosophy of its attainment. In secondary prevention, remedial actions cannot compensate for the mental anguish, physical pain, pretreatment disability, and the loss of productivity and contribution to the community thereby engendered. With primary prevention—the avoidance of disease itself—and the maintenance of health, however, none of these deleterious and impoverishing situations are encountered.

Paradoxically, the readily recognized advantages of primary prevention over arrest or cure

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Dr. Schuman is professor and head, division of epidemiology, School of Public Health, University of Minnesota, Minneapolis.

of disease have not been fully exploited. Generations have passed since mankind's observations and experiences led to the aphorism that "an ounce of prevention is worth a pound of cure," yet only lipservice has been given to this imperative in social behavior. It is deplorable that so much human waste has been tolerated when alternatives have been available. We cannot countenance the continuance of such dereliction in the face of evolving knowledge in primary prevention.

An exploration of some of the reasons for such dereliction could provide not rationalizations but understanding for correction of our deficiency in and support of primary prevention. The historical emergence of curative medicine for the immediacy of the need is readily apparent. The desperately ill, the dying man, the agonized victim, and the distraught in mind commanded and continue to command our attention and compassion; those who are not yet ill must wait, for we physicians are few and the emergent need is great. We have been trained, classically, to a practice of clinical triage and too little prevention. Remedial action constitutes almost the sole content of the physician's training and armamentarium. Related to, if not an integral part of, this continuing concept is the commonly held belief that our knowledge of primary prevention is grossly deficient, particularly in chronic diseases. This again is generated by the educational neglect in our schools of curative medicine.

Communicable Diseases

The individual accomplishments of primary prevention are well known, particularly in communicable diseases; for instance, the eradication of smallpox in the United States, the virtual eradication of bone tuberculosis and cholera, and the major declines in typhoid, diphtheria, plague, poliomyelitis, and pulmonary tuberculosis. Rarely recognized, however, is that even for some of these and other preventable infectious diseases there is a high residual incidence. Table 1 presents the reported incidence of selected communicable diseases for 1966; a large number of these are totally preventable and others can be reduced to much lower levels with means presently available.

Another disease entity which is totally preventable by the proper handling of streptococcal infections is rheumatic fever and its frequent sequel of chronic rheumatic heart disease. Yet in 1965, 4,908 cases of acute rheumatic fever were reported nationally. In addition to these new cases, 15,471 deaths from rheumatic fever and chronic rheumatic heart disease were reported. In fact, authorities frequently say that if all we know about rheumatic fever, diphtheria, and other infectious diseases were adequately applied, cardiovascular disease would virtually disappear as a cause of disability and death in the first 40 years of life.

Entities That Are Frequently Fatal

Concerning the control of chronic diseases, physicians, by omission, have been taught pessimism. True, that for many of these diseases our knowledge in regard to etiology is meager. The surface has barely been scratched, but these scratches have already released a torrent of new information applicable for primary prevention if we include not only those observations with etiological inference but also those of strong associative character.

Too frequently overlooked is that a number of chronic diseases, particularly those associated with certain occupations, have been preventable for some time and, relatively more recently, several occupational situations have been shown to contribute to the incidence of certain chronic diseases. Cancer of the lung among chromate workers, of the scrotum among chimney sweeps, of the bladder in workers with aniline dyes, and of the skin in outdoor workers under intense solar radiation are instances in point. Even more recently the etiological relationships between radionuclides and cancer of the lung in uranium miners and X-radiation and leukemia among radiologists have been demonstrated. These examples are valid as illustrations of the preventability of chronic diseases, but as health problems they are of relatively small magnitude.

For one disease, cancer of the lung, occupational risks contribute but a small portion of the total caseload. However, considering that the mortality rate from primary respiratory tract cancer, particularly lung cancer, almost doubled from 1950 to 1965, that by 1965 there were more than 52,000 deaths per year, and that more than 90 percent of the cases were due to ciga-

Table 1. Residual incidence of selected reported communicable diseases, 1966

Disease	Reported number of cases
Streptococcal sore throat and scarlet fever	427, 752
Gonorrhea	351, 738
Measles	204, 136
Syphilis	126, 573
Tuberculosis (newly reported active)	47, 767
Rubella	46, 975
Hepatitis (infectious and serum)	34, 356
Salmonellosis	16, 841
Shigellosis	11, 888
Pertussis	7, 717
Meningococcal infections	3, 381
Aseptic meningitis	3, 058
Amebiasis	2, 921
Encephalitis, primary	2, 121
Malaria	565
Typhoid fever	378
Rocky Mountain spotted fever	268
Brucellosis	262
Tetanus	235
Diphtheria	209
Tularemia	208
Trichinosis	115
Poliomyelitis	113
Leprosy	109

SOURCE: reference 1.

rette smoking, not only the direction but the feasibility of primary prevention becomes clear.

The same etiological agent or group of agents is also the most important of the causes of chronic bronchopulmonary disease and increases the risk of dying from chronic bronchitis and pulmonary emphysema. The growing importance of the problem and the dimension of the contribution to the nation's health which primary prevention could make can readily be seen from the increase in and magnitude of the mortality from these causes. In 15 years there was an eightfold increase in the number of deaths—from slightly more than 3,000 in 1950 to almost 24,000 in 1965—and a sixfold increase in the mortality rate from these causes (2.1 to 12.1 per 100,000).

When to lung, laryngeal, and lip cancer, chronic bronchitis and pulmonary emphysema are added the deaths from diseases strongly associated with tobacco smoking and for which the biomechanisms necessary for support of causal hypotheses seem to be emerging, the magnitude of the potential for primary prevention is encouraging. The diseases associated with tobacco use are shown in table 2. Mortality from these diseases comprised 47 percent of the total U.S. mortality in 1965.

If we consider only those diseases for which a causal relationship with tobacco use has been established or considered highly probable, they still account for 36 percent of the total. These are unrefined figures; they do not take into account the smoking segment of the population nor the contribution smoking makes to specific mortality. Thus, an examination of the data for the excess deaths among smokers over nonsmokers would be a better measure of the public health significance of this factor. From the prospective studies reviewed in the "Report of the Advisory Committee on Smoking and Health" in 1964 (*2*) and in the "Health Consequences of Smoking" in 1967 (*3*), approximately one-third of all deaths for men aged 35 to 60 would not have occurred if cigarette smokers had the same death rates as nonsmokers.

One could say facetiously, "but we all have to die sometime and from something." Irrespective of the mode of death, excess deaths are premature deaths—the excesses are primarily an earlier mortality—and, for the prime years of productivity, ages 45-49, they may reach an excess as high as 44 percent.

Information is gradually accumulating on the adjunctive and probably even synergistic role

Table 2. Mortality from selected chronic diseases related to tobacco smoking, 1965

Disease	Number of deaths
Causally related:	
Cancer of lung, bronchus, trachea	48, 483
Chronic bronchitis and emphysema	23, 432
Cancer of larynx	2, 629
Cancer of lip	172
Probably causally related:	
Coronary heart disease	559, 293
Cancer of bladder	8, 267
Cancer of buccal cavity and pharynx	6, 501
Cancer of esophagus	5, 542
Possibly causally related:	
Cerebrovascular disease	201, 057
Aortic aneurysm	10, 964
Total	866, 340
Total mortality, all causes	1, 828, 136

SOURCE: reference 4.

of community air pollution in the production of even greater excesses of mortality from respiratory cancer and chronic bronchopulmonary disease and of alcohol in the production of greater excesses of mouth, pharyngeal, and esophageal cancer and cirrhosis of the liver.

Lest we be too pre-occupied with death and forget the impact of illness on life's productivity and spiritual values, the revealing data on excess morbidity among smokers must be cited. These data were derived from special surveys within the framework of the National Health Survey of the National Center for Health Statistics. A summary of these findings (table 8) reveals that an appreciable excess of productivity loss occurs among smokers. This excess of significant morbidity among smokers, which diminishes their activity and hence productivity, peaks in the age group 45-64 and amounts to a 28 percent excess for each type of disability measured.

In other areas of potential primary prevention there are factors other than cigarette smoking in relation to the largest single disease problem in our country—coronary heart disease. Deaths from this entity contributed a minimum of 30.6 percent to the total mortality in 1965. Although cigarette smoking may be causally related, other risk factors besides smoking and age have been elicited epidemiologically which may be causes of coronary atherosclerosis.

High serum cholesterol levels and high blood pressure increase the risk for coronary artery disease manyfold. The relationship of serum cholesterol levels to dietary intake of saturated fatty acids has been well established. Evidence has been presented for the influence of dietary manipulation on the risk of coronary heart disease. We will have to be prepared for the early implementation of this finding as soon as larger scale studies confirm the initial observations. The impact of prevention of this increasingly occurring disease in our nation could be enormous.

Cerebrovascular disease which accounted for more than 200,000 deaths in 1965 (table 2) may well be amenable to the same primary prevention approaches as for coronary heart disease. Similar risk factors are involved—serum cholesterol (in persons under 50), hypertension, obesity, and cigarette smoking. A Public Health Service report stated that a number of the risk factors for stroke "are subject to correction or amelioration. Although direct evidence in the support of this contention is lacking, it seems altogether reasonable that many strokes could be postponed or averted by currently available countermeasures against salient risk factors particularly if the stroke-prone individual is identified early and preventive measures initiated promptly" (*6*).

Regarding infant mortality, a paradox exists in that this health index, which has been used as a measure of the progressiveness of health services in terms of availability and quality and of environmental control in a nation, is at a less-favorable level in the United States than in a number of other countries. At least 10 countries of Western Europe have better infant mortality experiences than ours. That other nations have achieved more favorable levels of infant health and survival automatically and forcefully implies a preventive potential which we have not yet tapped.

Disability

As I indicated previously, the prevention of early mortality and thus the prolongation of life is not the only goal of primary prevention—prevention of morbidity and thus provision for a well-adjusted and useful life is an even more important goal.

I have thus far dealt with disease entities for which death is a frequent and common sequel or for which the magnitude of the problem may be more or less derived from existing compulsory records such as reports of notifiable communicable diseases or death certificates. I must also mention acute conditions which are poorly or not at all reported and rarely lead to death, but which affect millions of persons and cause even more days of lost activity and pain or discomfort.

Through the National Health Survey, data are available on the common cold and other acute respiratory conditions including influenza. The estimated annual frequency of these conditions, for which a physician was consulted or which led to at least 1 day of activity restriction, was more than 240 million in 1966 and rep-

Table 3. Types of disability due to illness with excesses among smokers, aged 17 years and over, expressed as a percentage of the whole, United States, 1965

Type of disability	Total days lost	Excess days lost among smokers	Percent of total
Restricted activity	2, 360, 000, 000	306, 000, 000	13
Bed days	853, 000, 000	88, 000, 000	10
Work days lost	399, 000, 000	77, 000, 000	19

SOURCE: reference 5.

resented more than 332 million days of bed disability. These entities constituted 59 percent of all the acute illnesses or conditions and 48 percent of the bed-disability days. When the acute infectious and parasitic diseases are added, many of which are listed in table 1 and which actually equaled the number of accidental injuries sustained (48 million), these percentages rise to 71 and 69 respectively. The bed-disability days for the acute infectious diseases constituted approximately 40 percent of the bed-disability for all illness including chronic conditions. Again, the implications for prevention are clear.

Mental Health

The assessment of mental and emotional health is difficult, however, for neuroses and psychoses are not reportable and seldom lead to death. Furthermore, assessment of the problem by a count of beds occupied for mental illness is grossly misleading since any recent decline in such a count is probably the result of the use of ataraxic drugs rather than of a decline of illness. Yet, approximately half the hospital beds in the country are occupied by mentally ill persons.

Public mental institutions contain about a half million patients, and the National Health Survey estimated that 1,767,000 persons had mental and nervous conditions during the samplings of the population between July 1963 and June 1965. At best, this may well be an extremely minimal estimate because the counts were made only if major activities were limited and did not include persons in institutions, sanitariums, nursing homes, or homes for the aged. Furthermore, people tend to withhold information on mental conditions, and many cases are not diagnosed. This is admittedly an area of primitive understanding in terms of etiology, but it is highly probable that services for the emotionally disturbed could provide large returns in the prevention of more serious disturbances leading to mental illness.

Although death rates for homicide have declined by more than 35 percent in the past 30 years, our justifiable concern over the increasing rates of nonfatal criminal activity in our communities far exceeds concern for the phenomenon of suicide. Deaths by suicide have shown little tendency to decline in the past 30 years and certainly not at all in the past 20. The suicide rate is twice that of death by homicide. As a further comparison, in 1965 the suicide rate was as high—11.1 per 100,000—as the death rate for pulmonary emphysema—11.2 per 100,000 (International Statistical Classification 502.0, 527.1). Persons who commit suicide, however, are generally much younger than those who die of emphysema. Adequate psychiatric and social approaches are certainly indicated for primary prevention.

Much remains to be done for mental retardation also, but glimmers of hope for primary prevention appear in the demonstration of the role of dietary control in phenylketonuria.

Dental Caries

More than 25 years have passed since the demonstration of the inverse relationship between dental caries and the amount of fluorides in the water supply. Shortly thereafter, the experiment in Newburgh-Kingston, N.Y., proved that dental caries could be reduced 50 percent or more in the permanent teeth of children if their water supply were fluoridated. It is a sad commentary on the approaches that have been made in the prevention of this disease that more than two-thirds of the U.S. children are not being protected against caries through this simple means.

Child Health and Accidents

The gains in the life expectancy at birth or longevity during the past 50 years which we point to with pride were achieved primarily by saving children's lives, and this in turn by the reduction in the incidence of the great epidemic diseases. Little gains have occurred in the oldest age groups; proportionately, these have been far smaller. It is reasonable to assume therefore that rapid and telling gains in life expectancy can be achieved by increased efforts to protect the young against health hazards. One facet of the problem which I mentioned before is the far too high infant mortality rate.

The increased survival of infants into childhood and children into productive adulthood depends not only on vast improvement in infant mortality experience, but in the prevention of disability and death from accidents. It cannot be repeated often enough that accidents continue to be the principal cause of death in the United States for all age groups from 1 to 44 years. Table 4 illustrates the magnitude of mortality from accidents and reveals the contribution of motor vehicle accidents to the total, particularly in young adults. Although such accidents as falls and poisonings contribute to the bulk of childhood accident mortality, in the entire age range from 1 through 44 years deaths from motor vehicle accidents constituted 58 percent of the total deaths from all accidents in 1965.

The National Health Survey's statistics for July 1959 through June 1961 reveal an estimated 45 million injuries sustained each year, and approximately 3 million of these were due to moving motor vehicles. Whereas 41.9 percent of the persons with injuries from moving motor vehicles required 1 or more days of bed rest, only 21.4 percent of those injured in all other accidents required 1 or more days of bed rest. These data merely point up the reporting of many more less-serious accidents in the nonmotor vehicle group and attest to the lethality of the motor vehicle accident. Thus, the data on nonfatal injuries do not diminish, by any means, the importance of motor vehicle accidents. No one has thus far suggested that these are not totally preventable.

Potentials of Prevention

The examples I have cited not only constitute the bulk of the health problems besetting us today, but for most of these the hope of primary prevention is quite high. For several of these problems, existing preventive measures could be applied with great confidence for reduction of incidence if we but had the national will and the cooperation to do so. For others, certain strong associations have been demonstrated which are either modifying factors or determinants of the disease, and so they are worth manipulating before there is no longer any uncertainty as to their causal implications.

I have deliberately set forth disease problems for which primary prevention is a distinct reality or is highly probable. My appeal for primary prevention in no way disparages the continuing efforts which have recently led to regional medical programing and comprehensive health services planning.

I take no issue with the continuing fulfillment of the need for therapeutic medicine—for secondary prevention—which does indeed seek to alleviate pain, arrest or cure disease, and prevent disability and death. However, I do take

Table 4. Mortality from accidents, by type and rate per 100,000 population for selected ages, United States, 1965

Age group (years)	All accidents		Type of accident			
			Motor vehicle		All other	
	Number	Rate	Number	Rate	Number	Rate
1-4	5, 270	31. 8	1, 733	10. 5	3, 537	21. 3
5-14	7, 391	18. 7	3, 526	8. 9	3, 865	9. 8
15-24	18, 688	61. 7	13, 395	44. 2	5, 293	17. 5
25-44	22, 228	47. 8	12, 595	27. 1	9, 633	20. 7
Total, 1-44	53, 577	40. 3	31, 249	23. 5	22, 328	16. 8
All ages	108, 004	55. 7	49, 163	25. 4	58, 841	30. 4

Source: reference 4

issue with a way of life—a system—which minimizes, if not ignores, the potential for basic prevention of disease and suffering. I take no issue with the need to extend high-quality medical care to every citizen, although the methods proposed may be subject to criticism depending on our social viewpoint, but I do take issue with any system that ignores the preventive potential in its contact with society. I take no issue with the long overdue concept of integrating health services in the community so that there shall be as little waste through duplication as possible and no hiatus shall remain unfilled, but I do take issue with the minimal roles allotted to primary preventive procedures in such plans.

I firmly believe that in the long run human health, happiness, and useful longevity will be achieved at far less expense and with less suffering through primary prevention than through methods which seek to prolong the life of the ill. The ounce of prevention is figurative, for the cost of disability and death can be shown to far exceed a 16 to 1 ratio. We can never catch up with the problem until we begin to make inroads into the basic load of disease itself. Nor is the specter of a human population walking about with artificial hearts, kidneys, lungs, digestive tracts, and reproductive organs, and even computerized brain units, so wondrous to behold. The moral issues of these procedures may be far more profound than the addition of fluorides to a water supply which all will drink.

The psychosocial impact of a strictly curative or therapeutic philosophy also is not to be ignored. Through this philosophy's constant and demanded search for that elusive mistress—the cure—which is daily promised but rarely realized, a permissiveness is bred for our continuing transgressions on biology. It may even contribute to the behavior of our society which insists on perpetuating the paradox of "profits at any cost!"

Approaches to Primary Prevention

What, then, are the approaches to primary prevention? We may consider these to be operative in three areas of health activity: (*a*) personal health services, (*b*) environmental control, and (*c*) health education of the population.

Personal health services. An urgent need is the total reorganization of our thinking on the position and role of preventive medicine in the curriculums of our medical schools. So long as preventive medicine remains departmentalized instructionally as well as administratively, in my opinion the role and obligation of the American physician in true prevention of disease will neither be understood nor achieved. So long as preventive medicine remains only the tolerated partner, if that, in the medical school curriculum, the medical profession will not be indoctrinated with the concepts of preventive health services. This is an area of much needed experimentation and evaluation.

Some innovations via curriculum changes have been instituted in some schools recently, but these have been few and have come about as the result of the information explosion and not at all from a conceptualization of need for expansion of preventive health services. Innovations could readily include integration of prevention concepts and applications in phase-structured or track systems, or both, which envision a measure of specialization before the completion of the medical curriculum. In this latter regard, the development of cadres of physicians whose specialty is preventive health supervision within the structures of group, institutional, or community agency practice is certainly worthy of trial—a notion that is not new. Industrial medical programs have already adopted the concept of health supervision for prevention of disease. This concept needs to be extended to the total population so that medical care may truly become health care. A proper stimulus for the initiation of these innovations would be increased financial grants for their implementation.

Another compelling need in the medical curriculum is the exposure of all students to the concepts and contributions of the behavioral and social sciences to health care before they are rudely confronted with these problems and needs in practice and react, to the detriment of the patient, with antagonism. The role of these sciences in primary prevention as well as in disease supervision cannot be overestimated.

The concept of the proposed community health center, which should coordinate and integrate the activities of all health agencies in the community, certainly should embrace the services and practice of preventive health supervision. Through this mechanism, comprehensive health care as opposed to solely medical care can be achieved. It seems a logical place for the profession, community government, and the citizenry to come together for this goal.

Almost a generation ago the concept of multiphasic screening for chronic disease emerged—an outgrowth of experiences with casefinding for syphilis and tuberculosis. Its purpose was early detection of chronic disease, hopefully before symptoms appeared, so that arrest or cure could be more readily accomplished. Its applicational experiences during the past two decades have been good, although multiphasic screening was limited to diseases for which suitable and efficient tests were available.

Although directed toward existing but unknown disease, the elements of the screening approach can be directed readily toward elicitation of certain risk factors which may be the precursors of certain diseases and thus toward primary prevention also. The determination of blood pressure, smoking history, dietary pattern, and serum cholesterol and the simple determination of height and weight can provide enough information to screen the persons at high risk for coronary artery and cerebrovascular disease and provide them with preventive supervision and guidance. As research continues, screening tests for many other diseases will be developed, and it is not unreasonable to expect that many of them will elicit precursor abnormalities for further preventive applications.

Only modest funds have been expended for multiphasic screening activities, and these were primarily for demonstrations in too few areas. This failure of dissemination of an approach probably has been due to a combination of reasons—mainly apathy by official health agencies and the ignorance of its benefits and suspicion among the practicing profession. I suggest that every community health center incorporate a multiphasic screening program to attract apparently well persons in addition to sick ones and that every medical school incorporate student experience in such a screening program in its curriculum.

Although pediatricians and, to a somewhat smaller extent, obstetricians have applied more primary prevention in their practices than other medical specialists, benefits from their approaches have obviously accrued only to the patients who sought their services. Only through an extension of preventive practices to the entire population can we hope to expect some inroads on infant mortality, even though it is expected that universal application of certain environmental controls will contribute greatly to the reduction of the problem as it did in the first part of this century. In general, the problem of infant mortality is highly susceptible to preventive health supervision. The same basic concept which involves our rethinking of medical care as only part of health care would, for example, encompass the problem of emotional and mental health as well.

Environmental control. The basic concepts of eradication or isolation of environmental hazards were laid down long ago, and, for the most part, innovations in methods of application have stemmed from the peculiar characteristics of the newly emerging hazards as well as from technological developments in areas of old problems. Though not entirely synonymous, environmental control has implied community governmental control of a hazard which threatens most, if not always all, members of the community. Our historical governmental regulations of water supplies, sewage disposal, milk supplies, and, to an inadequate extent, other food sources are instances in point. Recent governmental regulatory intervention in environmental hazards, long standing or newly emerging, include the as yet embryonic control of water pollution, air pollution, and radiological hazards. Drug control, although also historic, only recently has been given new directions toward its goals of prevention of therapeutic misadventure and economic waste from the application of useless drugs.

Although normally taking the pattern, both in the community as a whole or in industry as a segment, of removal or isolation of the specific hazard, innovations in basic philosophy, however, have occurred at times. The addition of iodine to table salt for prevention of colloid goiter, the addition of fluorides to water for prevention of dental caries, and the fortification of foods are certainly departures from the basic pattern. These, however, are precedents for the future.

Thus, as a second, but certainly not secondary approach to primary prevention, environmental control provides a vast promise. Hopefully, self-regulation is not a totally dead issue, but the experiences of the past, and particularly the immediate past, with the tobacco industry engenders impatience with self-regulation for the control of hazards. We turn instead to the only alternative—governmental regulation—for it is our lives and health which are at stake and which should not be bargained for.

Governmental control of the environment may take on prohibitive or regulatory functions (including setting of standards) or both. In water and air pollution, including the discharge of radioactive wastes into both media, continued and even more aggressive control by prohibition of some and regulation of other effluent practices is indicated. Continued aggressive action is certainly needed in drug control, including the control of addictive and psychedelic drugs. Standard setting and engineering control for the safety of motor vehicles will have to be expanded, and serious consideration must be given in the immediate future to either mass public transit to cut down the needs for private vehicle use or to truly automated control of private vehicles.

Another example of environmental control by government, both Federal and local, is the necessary extension and strengthening of regulatory control of the food processing industries, particularly meat and poultry.

Since prohibition applied to a person's habits is doomed to failure, as was evidenced by alcohol prohibition, indirect forms of prohibition or regulation became necessary. In the consideration of either tobacco or alcohol, I believe control on a national basis will have to include prohibition of all advertisement of either of these environmental hazards; for tobacco, rigid local enforcement of sales to minors, as conducted to a greater extent for alcohol, is needed.

In the event that dietary manipulation is proved practical as a preventive of coronary heart disease and possibly even cerebrovascular disease, changes in food fat composition by industry would no more be out of order than the iodizing of salt or the fluoridation of water supplies.

For years, public health proponents have recognized the impact of poverty, housing, and other social factors on health. The role of crowding and socioeconomic status in the production of rheumatic fever, as one example among many, was established many years ago. These social factors must be considered as much a part of the environment as chemical, physical, and biological hazards. Recently, certain sociological concepts of the community, such as anomie, have emerged and these too will have to be considered in terms of primary prevention of disease or better still the promotion of health, particularly emotional and mental health.

Health education. As an approach to primary prevention, health education is probably among the most difficult. Education for health has a twofold purpose: (*a*) education for personal health which is necessary to bring the individual to preventive health services of any type and (*b*) education for community action in health which is the only way to guarantee that the community will safeguard itself against environmental hazards by legal regulatory actions and will provide personal health services. As an educator, I have faith in the process which must go on inexorably, if slowly.

Conclusion

The illustrations of health problems for primary prevention I have presented do constitute the important areas of ill health in our society today. The suggestions for their solution are but fragmentary and representative of a variety of methods and modalities which could be applied. Throughout this commentary are not-so-veiled suggestions of necessary research, epidemiologic in character, not only on further etiological relationships and determinants of disease, but on program and educational applications. Furthermore, a careful and deliberate assessment of the problems in specific detail for practical priorities in the achievement of prevention must be undertaken.

For these reasons, I would urge the establishment of a commission to study the total problem of prevention, its perspectives, the pragmatical areas of preventive capabilities of our society, the ways and means of their application, the augmentation of teaching in this field, and the delineation of the areas of necessary and immediate intensification of research. The justification for such study is simply that prevention of disease is the ethical imperative of our social order.

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Tearsheet Requests

Leonard M. Schuman, M.D., Division of Epidemiology, University of Minnesota School of Public Health, 1325 Mayo Memorial Building, Minneapolis, Minn. 55455

Reorganization of DHEW Programs for Mothers and Children

Recent organizational changes which will strengthen Department of Health, Education, and Welfare programs affecting child welfare, social services, and maternal and child health care are as follows.

- The Children's Bureau has been moved from the Social and Rehabilitation Service (SRS) to the Office of the Secretary, where it becomes part of the new Office of Child Development (OCD). The Bureau will maintain its role of leadership and coordination of child and parent programs throughout the Department. It will also continue to investigate and report on all matters pertaining to the welfare of children, under the 1912 act which created it. The OCD will report to the Secretary through Assistant Secretary for Administration James Farmer.

With the move, OCD now consists of three major elements: the Children's Bureau, Bureau of Head Start and Child Development, and Bureau of Program Development and Resources.

- A Community Services Administration has been established in SRS to consolidate the administration of social service programs from children and adults. These include programs located previously in the Children's Bureau and in other SRS agencies. It will operate as a single point of responsibility at the Federal level for social services offered through State and local welfare agencies. Stephen P. Simonds has been designated as acting commissioner of the Community Services Administration.

- Health programs administered by the Children's Bureau have been transferred to the Health Services and Mental Health Administration (HSMHA) where they will comprise a new organizational unit, the Maternal and Child Health Service. Dr. Arthur J. Lesser has been named acting director. Programs included are for maternal and child health services, crippled children, maternity and infant care, and health of school and preschool children.

The National Center for Family Planning Services, whose acting director is Dr. Stanley C. Scheyer, has been established within HSMHA. The Center will develop family planning programs for DHEW, mesh them together with other Federal efforts, and administer family planning project grant activities for HSMHA. It will also function as a clearinghouse for the collection, organization, and dissemination of family planning information.

Epidemiology of Smoking Related Diseases Which Physicians Encounter in Their Office Practice*

*Leonard M. Schuman, M.D.***

The practicing physician can no more ignore the smoking habits of his patients than he can avoid the process of diagnosing the illness that brings that patient to him. Even if there were no associations, causal or contributory, between smoking and disease, the average practitioner would find that over half (51.0 per cent) of his men patients and one-third (33.2 per cent) of his women patients aged 17 years and over are regular cigarette smokers.[1] This may be compared with the prevalence of cigarette smoking in the United States as derived from the Current Population Survey for February 1955.[2] In that year, 49.8 per cent of men and 23.6 per cent of women aged 18 years and over were regular cigarette smokers. Thus, in one decade, women smokers increased approximately 50 per cent with but a slight increase for the men. The women can thus be seen to be rapidly approaching the men in prevalence of the cigarette smoking habit, and for both sexes, this habit constitutes one of the most common exposures to an environmental hazard.

These smoking prevalence data represent the patterns for the population irrespective of illness. When one considers the high degrees of association between cigarette smoking and mortality and morbidity from such diseases as lung cancer, chronic bronchitis and emphysema, cancer of the larynx, coronary heart disease, cancer of the bladder and esophagus, and cerebrovascular disease and between other forms of tobacco use and cancer of the buccal cavity, it is logical to conclude that an even greater proportion of all patients seen by the practicing physician will present a history of current regular smoking.

The gravity of the situation which we now face can be expressed in several ways. In 1965, of the 1,828,136 deaths from all causes, 866,340 (47 per cent) were from diseases associated with tobacco use (Table 1). Even if we restrict our attention to those diseases for which a causal relationship with tobacco use has been established or considered highly probable, we are still dealing with 36 per cent of the total mortality in the United States. Furthermore, from the prospective studies of U.S. veterans, British physicians, Canadian pensioners and the Hammond study of men and women in 25 states which have yielded additional data since 1964,[3] an estimate can be made that *one-third* of all deaths for men aged 35 to 59 would not have occurred if cigarette smokers had the same death rates as nonsmokers. Thus, these excess deaths were not confined to the aged. Irrespective of mode of death, these excesses were premature deaths—an earlier mortality—and, for the prime years of life's productivity, from ages 45 to 49, reached an excess as high as 44 per cent in one study (Hammond's). It is of related interest that it is in this age group for both sexes that today we find the highest proportions of heavy smokers.[1]

In terms of morbidity, and irrespective of types of illness, individuals who have smoked have a significant excess of disabling illness. The impact of such illness as measured by the National Health

Table 1.—***Mortality from Selected Chronic Diseases Related to Tobacco Smoking, 1965.***

Disease	No. of Deaths
A. *Causally Related:*	
Cancer of lung, bronchus, trachea	48,483
Chronic bronchitis and emphysema	23,432
Cancer of larynx	2,629
Cancer of lip	172
B. *Probably Causally Related:*	
Coronary heart disease	559,293
Cancer of bladder	8,267
Cancer of buccal cavity and pharynx	6,501
Cancer of esophagus	5,542
C. *Possibly Causally Related:*	
Cerebrovascular disease	201,057
Aortic aneurysm	10,964
Total	866,340
Total Mortality, all causes	1,828,136

Source: Vital Statistics of the U. S., 1965, Vol II, Mortality, Part A.

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*Presented at the National Forum on Office Management of Smoking Problems, American College of Chest Physicians, April 11, 1968.

**Professor and Head, Division of Epidemiology, School of Public Health, College of Medical Sciences, University of Minnesota, Minneapolis, Minnesota.

Survey[1] provides us with another perspective of the magnitude of the problem. Thirteen per cent of all days lost by restricted activity among the U.S. population aged 17 and over in 1965 represents the excess lost among smokers. Smokers also showed an excess of 10 per cent of all days lost by confinement to bed and a 19 per cent excess of work days lost. Though these percentages seem small, they actually represent excesses of 306,000,000 man-days lost by restricted activity, 88,000,000 man-days lost by confinement to bed; and 77,000,000 man-days lost from work. Here also, as had repeatedly been demonstrated for mortality in retrospective and, particularly, prospective studies,[4] these excesses, or increased risks for morbidity, were dose-dependent, i.e. the excesses of days lost due to disabling illness among smokers as compared to non-smokers increased as the number of cigarettes smoked per day increased. This was true for both men and women with the women showing consistently higher excesses in rates of restricted-activity days and of days of bed-disability although the excess of days of work loss was apparently the same for both sexes. Even here, the excess over non-smokers and the rising gradient of this excess with dosage of cigarettes was quite apparent.[1,3] The age at which these excesses of disability days peaked was the 45–64 years age group for men in which age-group are found the heaviest smokers in the entire life span. For women, the peak age of excess of disability days was in the age group 17–44 years. This earlier peak can be explained by the higher proportion of smokers in this age group.

In the National Health Survey, one or more chronic conditions were reported more frequently by both men and women smokers of cigarettes than by non-smokers. It has been estimated[3] that for the U.S. population aged 17 and over, there are now 11,000,000 more cases of chronic illness annually than there would be if all of the population had the same chronic disease morbidity (prevalence) rate as those who had never smoked cigarettes. A large proportion of the chronic illnesses reported in the survey are accounted for by such conditions as chronic bronchitis and emphysema, heart conditions, peptic ulcers and sinusitis. Here also, the frequency of reporting of such chronic conditions increased as the daily amount of cigarettes consumed increased.

It will be of value to examine the individual diseases related to smoking that apparently contribute to these excesses of mortality and morbidity and which, obviously, will constitute a great share of the average physician's practice. Probably of greatest importance among the smoking-related diseases, since it produces the bulk of the mortality among them, is *coronary heart disease*. Although the Report of the Advisory Committee to the Surgeon General did not find the data in 1964 adequate for the judgment of causality in the association between cigarette smoking and coronary heart disease, evidences in favor of this interpretation of the association continue to accumulate. Such evidences include the extension of the previous prospective studies, additional data clarifying adjunctive risks, morbidity data from the National Health Survey and experimental studies providing clues to the biomechanisms involved.[3]

Coronary heart disease was the cause of death recorded for 550,293 persons in 1965 for a mortality rate of 288 per 100,000.[5] These deaths accounted for 30.6 per cent of all the mortality in the United States that year. From more recent data in the major prospective studies, the evidence is clear that although the *excess risk* of coronary heart disease among smokers of cigarettes as expressed by a mortality ratio is greatest for both men and women in the 45–54 year age group and diminishes with age, the actual *number* of excess deaths per 100,000 smokers increases with increasing age. This can be interpreted to mean that although cigarette smoking tends to have a lesser role in coronary heart disease deaths with increasing age, its impact remains great.

More recent data on coronary morbidity also tend to support the findings from the prospective studies of mortality. The National Health Survey[1] in 1965 recorded age-specific prevalence rates of 2.4 per cent for men aged 45–64 years, 3.6 per cent for men aged 65 years and over, 1.0 per cent for women in the 45–64 year age group and 2.6 per cent for women aged 65 years and over. Among both men and women, persons who had ever smoked and current smokers had significantly higher prevalence rates than non-smokers. The excesses of rates for smokers were greater for the age group 45–64 years than for the group 65 years and over. Thus again it will be noted that the majority of patients with coronary heart disease will be cigarette smokers. *Incidence* figures for coronary heart disease are also available from the Framingham Study and, if this community is at all representative of most communities in the United States, these data provide us with average estimates of expectancy. In Framingham, from a 12-year experience through 1966, the incidence rate of coronary heart disease among cigarette smoking men aged 35–44 was 4.1 per 1000, or 2.9 times the rate among non-smokers of that age, among 45-54 year olds it was 11.1 per 1000, or 2.4 times the non-smoker rate; and among 55-64 year olds, 25.4 per 1000, or 1.6 times the non-smoker rate. The actual excess of the rates among smokers as compared to non-smokers increases with age from 2.7 per 1000 to 9.2 per 1000, but the rate of increase in incidence rates among non-smokers is far greater indicating first that although smoking may contribute to this disease at all age levels, its greatest contribution is at the youngest level and, secondly, that other risk factors contribute more at the higher ages.

More evidence of an association between cigarette smoking and cerebrovascular disease has appeared since the 1964 Report to the Surgeon General.[4] With 201,057 deaths attributed to this cause in 1965, representing approximately 11 per cent of the total U.S. mortality that year, this entity is also relatively common to the physician's practice. Although deaths of men from cerebrovascular disease exceed those of women, the excess mortality from this cause among cigarette smokers as compared to non-smokers (and hence the mortality ratios) is greater among women than men. Again, as in coronary heart disease, the impact of cigarette smoking on this disease appears to be greater in the 45-54 year age groups than in those over 75 years, with a smooth gradient of decline of relative risk with increasing age. This phenomenon in both these disease entities emphasizes the association of cigarette smoking with far greater earlier mortality.

The disease which is by far the most strongly causally related to cigarette smoking is lung cancer. This disease, which continues to show an alarmingly exponential rise of mortality and for which presently available early diagnostic and therapeutic methods leave much to be desired, claimed 48,483 lives in 1965 or 2.6 per cent of the total U.S. mortality. Both men and women are experiencing this increase in mortality which has doubled in rate since 1950, being 25 per 100,000 in 1965. Since 1960, the population of women has been experiencing a relatively greater increase in lung cancer mortality rate belying the commonly held lay belief that women are not susceptible to the carcinogenic action of cigarette smoke. At the present time, the mortality rate among men is over 41 per 100,000, while in women the rate is over seven per 100,000 or approximately one-sixth of the rate for men. The increase in lung cancer mortality in both sexes tend to parallel the corresponding increases in rates of cigarette consumption among them. Mortality from lung cancer has also risen faster in the non-white population than in the white, so that today the rates for whites and non-whites, though differing by sex, are equal.[4]

The data from the seven prospective studies and several of the retrospective studies revealed a 9 to 11-fold risk of lung cancer among men cigarette smokers as compared to non-smokers. The rates in the Hammond study were 87 per 100,000 among smokers as compared to 11 for non-smokers at ages 45-64. The rates for women were 15 as opposed to 7 per 100,000 in this age group. In the 65-79 year age group, the rates were 262 as opposed to 23 for men and 30 as opposed to 17 for women. It is these considerably higher mortality ratios for lung cancer in smokers that places this entity second to coronary heart disease as a producer of excess mortality among cigarette smokers even though lung cancer accounts for only one-eleventh as many deaths as are attributed to coronary heart disease. Whereas coronary heart disease accounted for approximately 44 per cent of the excess deaths among cigarette smokers, lung cancer accounted for over 17 per cent of the excess.[4]

In lung cancer, we find one of the most precise dose-response relationships with cigarette smoking in terms of the current number of cigarettes smoked per day, the degree of inhalation, age when smoking was started and number of years smoking had been discontinued. It is in this smoking-related disease that we find concrete evidence of the benefits of discontinuance or reduction of the smoking habit. In the ten-year period 1954 to 1964, whereas the amount of smoking in the general population of England and Wales had not decreased and the lung cancer mortality had increased by 25 per cent, the British physicians in the Doll study, among whom there was a substantial drop in cigarette smoking, have now experienced a 30 per cent decline in mortality from this disease.[3] This should be sufficient stimulus for the physician to encourage all his patients to change their smoking habits.

Chronic bronchopulmonary diseases were causally implicated in the 1964 Advisory Committee Report to the Surgeon General. The evidence was deemed adequate for a causal relationship between cigarette smoking and chronic bronchitis and it was strongly implicated as increasing the risk of death from pulmonary emphysema. Despite the recent attempts at more precise definitions of chronic bronchitis and pulmonary emphysema, the status of such definitions over the immediate past in our own country and those in other countries makes trends and comparisons difficult if we attempt to separate the two entities. Thus, mortality and morbidity analyses are better applied to a combination of the two—chronic bronchitis and/or emphysema. As a problem in *mortality*, chronic bronchitis and/or emphysema presently claim half as many deaths as does lung cancer. However, in the 16-year period, 1950–65, deaths from these two non-neoplastic pulmonary diseases have increased over six-fold from 3,157 deaths reported in 1950 to 23,432 reported in 1965.[6,5] This increase has been greater than the similarly appalling toll from lung cancer. The most dramatic rise has been for the emphysema component for which the age-adjusted mortality rates rose from 1.3 per 100,000 in 1950 to almost 13.0 in 1965. Deaths from chronic bronchitis rose less spectacularly, only doubling in the same period. Although some of these increases must represent diagnostic improvement, it is difficult to ascertain the degree to which this operated. The causal relationship with cigarette smoking and the use of cigarettes in this period would suggest that a major part of this rise in mortality must be real.

Next to lung cancer, chronic bronchitis and pulmonary emphysema were most highly associated with current cigarette smoking as measured by mortality ratios. In these diseases the risk for the cigarette smoker was more than six times the risk for the non-smoker as indicated in the seven prospective studies.[4] Men had somewhat higher mortality ratios than women for both middle and upper age-groups in the Hammond study.[3] Here, as in lung cancer, the dose-response follows a very distinct gradient with increasing daily consumption of cigarettes. This was noted in each of the studies with virtually no discontinuities on the dose scale. In the study of U.S. veterans, men non-smokers had a mortality rate of two per 100,000 in the 55–64 year age-group, whereas smokers of cigarettes had rates ranging from 12 per 100,000 for under 10 cigarettes a day to 39 per 100,000 for 40 or more cigarettes a day with an average of 29 per 100,000. In the 65–74 year age-group, the mortality rate for non-smokers was 10 per 100,000 and for smokers of two packs a day or more this rate was 322 with an average of 113 per 100,000 smokers of any quantity of cigarettes.

As producers of *morbidity*, chronic bronchitis and pulmonary emphysema are definitely more efficient than coronary heart disease. In the National Health Survey for 1965[1] 1.9 per cent of the men and 2.0 per cent of the women reported chronic bronchitis and/or emphysema as compared to 1.4 per cent and 0.7 per cent, respectively, reporting coronary heart disease. Men and women smokers had more than twice the morbidity of the non-smokers and in the older age groups this ratio was even higher.

The other causally related cancers of the larynx and lip, the probably causally related cancers of the bladder, buccal cavity, pharynx and esophagus, as well as the possibly causally related non-syphilitic aortic aneurysm, accounted for a total of 34,075 deaths in 1965.[5] This mortality load falls between that for lung cancer and chronic bronchitis and emphysema.

The data on smoking-related diseases of man are thus compelling. They are more than sufficient for the practicing physician to be seriously concerned for the vast majority of his patients, for it is obvious that regardless of the presenting complaint of the patient, the role of prevention of the diseases discussed is clear and its institution imperative. Nor will the status quo prevail, for not only are the mortality rates for lung cancer, chronic bronchitis and emphysema increasing, but also those for coronary heart disease, cancer of the esophagus and, for the woman, gastric ulcer. The experiences of the British physicians prospective study with the substantial decreases in mortality from not only lung cancer, but also chronic bronchitis, as their smoking decreased after the preliminary reports of the study, not only strengthen the conclusion of a causal relationship but, more important, provide the evidence that prevention is indeed possible.

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Reprint requests: Dr. Schuman, 1158 Mayo Building, University of Minnesota, Minneapolis 55455.

ASH
ACTION ON SMOKING AND HEALTH
2013 H St., N.W. • Washington D.C. 20006 • (202) 659-4310

TESTIMONY OF ACTION ON SMOKING AND HEALTH (ASH), BY ITS EXECUTIVE
DIRECTOR AND CHIEF COUNSEL, JOHN F. BANZHAF III, BEFORE THE HOUSE
SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENT, CHAIRED BY THE
HONORABLE HENRY A. WAXMAN, ON THE "COMPREHENSIVE SMOKING
PREVENTION EDUCATION ACT OF 1981," (H.R. 4957)
Submitted March 22, 1982

I am happy to appear before you on behalf of Action on Smoking and Health (ASH) to strongly support the "Comprehensive Smoking Prevention Education Act of 1981." As you may know, Action on Smoking and Health is a national nonprofit organization which serves as the legal action arm of the antismoking community. In this capacity it has been directly involved in virtually all major actions concerning cigarette smoking and advertising.

For example, in my individual capacity I filed the complaint at the Federal Communications Commission which led to the requirement that stations make free time available for antismoking messages under the Fairness Doctrine. Shortly thereafter ASH was formed and played a major role in upholding the Congressionally-imposed ban on cigarette advertising. ASH was also instrumental in forcing "little cigar" ads off the air and in persuading the Federal Trade Commission to file complaints against the tobacco industry concerning various cigarette advertisements.

In my individual capacity as a Professor of Law at the National Law Center of the George Washington University I was able to persuade the Federal Trade Commission to adopt corrective advertising as a weapon against deceptive ads. Since that time I have participated in numerous proceedings concerning deceptive advertising at the Commission, including a major fact-finding proceeding concerning the permanent effects of advertising.

On the basis of this experience it is my judgment that the requirement of stronger, clearer, more specific health warnings is a necessary and long-overdue step. This, coupled with the idea of changing, or "rotating," warnings will do much to make them more effective and to bring them forcefully to the public's attention.

Because the need for these warnings has been amply demonstrated in other testimony and in many studies by the Federal Trade Commission and other agencies, I will not dwell on this aspect. However, I think it is important for the Congress to appreciate the depths to which the tobacco industry will descend to try to lure young people into smoking and to negate or otherwise distract their attention from the serious health problems it presents. To dramatize this, I am attaching, as part of my testimony, a portion of a confidential report prepared by the Staff of the Federal Trade Commission and -- for reasons one may well conjecture -- not made available to Congress or the public.

The report demonstrates many things. First, the industry apparently has nothing but contempt for people who use its product or people who might be persuaded to do so. For example, a tobacco industry report at page 3-16 of the Federal Trade Commission's confidential document says,

> Then, the smokers have to face the fact that they are
> illogical, irrational and stupid ...
>
> * * * * * * *
>
> ... smokers don't like to be reminded of the fact that
> they are illogical and irrational.

LEGAL ACTION AND EDUCATION ON THE HAZARDS OF SMOKING • PROTECTING THE RIGHTS OF THE NONSMOKING MAJORITY

The report also demonstrates that the tobacco industry actively encourages smoking by young people and actively designs campaigns to encourage them to begin smoking. For example, among the strategies for attracting young "starters" to cigarette smoking are the following:

> Present the cigarette as one of a few initiations into the adult world
>
> Present the cigarette as part of the illicit pleasure category of products and activities.
>
> * * * * * * *
>
> To the best of your ability, (considering some legal constraints), relate the cigarette to 'pot', wine, beer, sex, etc.

Third, this confidential document makes it abundantly clear that the tobacco companies use a variety of means to detract from and undermine the health warning on cigarettes. On this point I think the report speaks most forcefully for itself.

In addition to including this report as a part of ASH's testimony, Action on Smoking and Health would like to respectfully request and suggest that the Committee demand from the Federal Trade Commission the various documents cited in the report and place them, also, in the record, so that Members of Congress may be fully advised on the tobacco industry's strategy and techniques and the effectiveness of their advertisements prior to a vote on this issue.

THIS DOCUMENT CONTAINS
CONFIDENTIAL INFORMATION

FEDERAL TRADE COMMISSION

STAFF REPORT ON THE CIGARETTE ADVERTISING INVESTIGATION

BY:

Matthew L. Myers
Program Advisor

Craig Iscoe
Carol Jennings
William Lenox
Eleanor Minsky
Andrew Sacks

APPROVED:

Collot Guerard
Deputy Assistant Director
Division of Advertising Practices

Wallace S. Snyder
Assistant Director
Division of Advertising Practices

CONCUR:

James Sneed
Director
Bureau of Consumer Protection

May, 1981

> explicit and varied. Their constant repetition in advertising which reaches vast numbers of Americans of all ages must be viewed as significantly contributing to the portrayal of the desirability of smoking.[30]

Thus, the dominant themes of cigarette advertising are that smoking is associated with youthful vigor, good health, good looks and personal, social and professional acceptance and success, and that it is compatible with a wide range of athletic and healthful activities. One theme is conspicuously absent from all cigarette ads. Although these ads contain the required general warning, they make no mention of the numerous and specific adverse health consequences of using the advertised product.[31]

IV. ADVERTISING THEMES AS DESCRIBED BY THE MANUFACTURERS' MARKETING PLANS

The cigarette companies' documents reinforce the findings of the staff about the themes of cigarette advertising. For example, R.J. Reynolds' 1977 marketing plan for Salem states explicitly that:

> Motivational research has identified the phenomenon of image projection as a highly motivating force. Therefore, through the association of SALEM, and its brand styles with emulatable personalities and situational elements that are compatible with the aspirations and lifestyles of contemporary young adults, this important target segment will be attracted to the brand. Importantly, older smokers also relate

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[30] 1964 Cigarette Rule, _supra_, 20 Fed. Reg. at 8342.

[31] To the best of our knowledge, cigarette companies never have provided health information, other than the required warning and "tar" and nicotine figures, in their advertisements.

> favorably to this personality type; therefore, reinforcement of the current franchise is simultaneously achieved.[32]

The same marketing plan indicates that a primary theme for the promotion of Salem has been to associate the cigarette with the lifestyle of "young adult males" who are "masculine, contemporary, confident, self-assured, daring/adventurous, mature."[33] Marketing plans for other cigarettes are similar. A Doral campaign sought to project the image of "an independent, self-reliant, self-confident, take-charge kind of person."[34] A Winston man was projected as "a man's man who is strong, vigorous, confident, experienced, mature."[35]

Liggett & Myers' documents show that their Lark ads picturing a balloon high above land sought to give the consumer an association with "lightness and exhilaration."[36] L&M's campaign planned to position Lark as a "youthful, contemporary brand that satisfies the lifestyles of the modern smoking public." Its ads emphasize "moments of post-tension and relaxation."[37] The Eve smoker has been portrayed as a "sophisticated, up-to-date, youthful and active woman who seems to have distinct ideas about what she wants."[38]

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[32] Document A900022 - "Salem 1977 Annual Marketing Plan."

[33] Id.

[34] Document A900003 - "RJR Statement of Business 1977 - Doral Cigarettes."

[35] Document A900001 - "Winston King 1978 Marketing Plan."

[36] Document A900230 - "1974 Lark Annual Marketing Plan."

V. EFFORTS OF SOME CIGARETTE ADVERTISEMENTS TO DIVERT ATTENTION AWAY FROM THE HEALTH HAZARDS OF SMOKING

Many cigarette advertising techniques appear to denigrate or undercut the health warning. Information obtained from subpoenaed documents indicates that, at least in the case of several advertising campaigns, these techniques have been carefully planned. For example, documents from Brown & Williamson (B&W) and one of its advertising agencies, Ted Bates and Company, Inc., set forth the development of an advertising strategy for Viceroy cigarettes designed to suppress or minimize public concern about the health effects of smoking.

The documents show that, at the request of Ted Bates, a marketing and research firm conducted a number of focus group interviews on the subject of smoking in order to assist the ad agency in developing a marketable image for Viceroy cigarettes.[39] The final report summarizing the results of this research asserts that many smokers perceive the smoking habit as a "dirty" and dangerous one engaged in only by "very stupid people."[40] The report concludes:

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[37] Document A900251 - "1973 L&M Marketing Plan."

[38] Document A900245 - "1974 Eve Portfolio Test."

[39] Document A011345 - "An Action-Oriented Research Program For Discovering And Creating The Best Possible Image For Viceroy Cigarettes," prepared for Ted Bates Advertising in March 1975 by N. Kennan, Marketing and Research Counselors, Inc.

> Thus, the smokers have to face the fact that they are illogical, irrational and stupid. People find it hard to go throughout life with such negative presentation and evaluation of self. The saviours are the rationalisation and the repression that end up and result in a defense mechanism that, as many of the defense mechanisms we use, has its own 'logic', its own rationale.
>
> * * * * * * * *
>
> Thus, smokers don't like to be reminded of the fact that they are illogical and irrational. They don't want to be reminded by either direct or indirect manner.[41]

The report proceeds to describe the elements of a good cigarette advertising campaign, in light of its findings, in a chapter entitled, "How To Reduce Objections To A Cigarette." The basic premise of the report's recommendations is that since there "are not any real, absolute, positive qualities and attributes in a cigarette," the most effective advertising is designed to "reduce objections"[42] to the product by presenting a picture or situation ambiguous enough to provide smokers with a rationale for their behavior and a means of repressing their health concerns about smoking. To provide a rationale for smoking, the ad must project the image that cigarettes provide the smoker with social acceptance, an acceptable means of rewarding himself or herself, a stimulant, a tranquilizer, a better self-image, etc. With regard to health issues, the report recommends: "Start out from the basic assumption that cigarette smoking is dangerous to your health - try to go around it in an elegant manner but don't try to fight it - it's a losing war."[43]

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[40] Document A901268 - May 26, 1975 "What Have We Learned From People? A Conceptual Summarization of 18 Focus Group Interviews On The Subject Of Smoking."

[41] Id. at 2, 3 (emphasis in original).

[42] Id. at 12 (emphasis in original).

One chapter of the report describes how the company can introduce "starters" to the Viceroy brand, a discussion which focuses almost exclusively on how to persuade young people to smoke. The report asserts:

> For the young smoker, the cigarette is not yet an integral part of life, of day-to-day life, in spite of the fact that they try to project the image of a regular, run-of-the-mill smoker. For them, a cigarette, and the whole smoking process, is part of the illicit pleasure category...In the young smoker's mind a cigarette falls into the same category with wine, beer, shaving, wearing a bra (or purposely not wearing one), declaration of independence and striving for self-identity. For the young starter, a cigarette is associated with introduction to sex life, with courtship, with smoking 'pot' and keeping late studying hours.[44]

The chapter then recommends a strategy for attracting young "starters" to cigarette smoking:

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[43] Id. at 17.

[44] Id. at 29-30 (emphasis in original).

Thus, an attempt to reach young smokers, starters, should be based, among others, on the following major parameters:

- Present the cigarette as one of a few initiations into the adult world.
- Present the cigarette as part of the illicit pleasure category of products and activities.

* * * * * * * *

- In your ads create a situation taken from the day-to-day life of the young smoker but in an elegant manner have this situation touch on the basic symbols of the growing-up, maturity process.
- To the best of your ability, (considering some legal constraints), relate the cigarette to 'pot', wine, beer, sex, etc.
- Don't communicate health or health-related points.[45]

B&W adopted many of the ideas contained in this report in the development of a Viceroy advertising campaign. Thus, in a document entitled, "Viceroy Strategy," B&W notes repeatedly that its advertising campaign must provide consumers with a rationalization for smoking and a "means of repressing their health concerns about smoking a full flavor Viceroy."[46] The following excerpts from "Viceroy Strategy" are representative and indicate that in B&W's view, the other cigarette companies also have developed advertising strategies designed to cause repression of consumer health concerns about smoking:

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[45] Id. at 31.

[46] Document A015538 - "Viceroy Strategy," March 3, 1976, V.C. Broach, Group Project Manager, B&W (emphasis in original).

> Full flavor smokers perceive cigarette smoking as dangerous to their health...Given their awareness of the smoking and health situation, they are faced with the fact that they are behaving illogically. They respond to this inconsistency by providing themselves with either a rationalization for smoking, or, by repressing their perceptions of the possible dangers involved. To date, major full flavor brands have either consciously or unconsciously 'coped' with the smoking and health issues in advertising by appealing to repression. [emphasis added.]
>
> * * * * * * * * *
>
> The marketing efforts must cope with consumers' attitudes about smoking and health, either providing them a rationale for smoking a full flavor VICEROY or providing a means of repressing their concerns about smoking a full flavor VICEROY. [emphasis in original.]
>
> * * * * * * * * *
>
> Advertising Objective - To communicate effectively that VICEROY is a satisfying flavorful cigarette which young adult smokers enjoy, by providing them a rationalization for smoking, or, a repression of the health concern they appear to need.

B&W then describes its plan to accomplish its advertising objective. Three advertising strategies would be used:

> 1. The 'satisfaction' campaign provides a rationalization: VICEROY is so satisfying that smokers can smoke fewer cigarettes and still receive the satisfaction they want....
> 2. The 'tension release' campaign provides a rationalization: VICEROY'S satisfying flavor can help the smoker in a tense situation....
> 3. The 'feels good' campaign appeals to the smoker by repressing the concerns he may have about smoking by justification: If it feels good, do it; if it

feels good, smoke it....[47]

B&W documents also show that it translated the advice on how to attract young "starters" into an advertising campaign featuring young adults in situations that the vast majority of young people probably would experience and in situations demonstrating adherence to a "free and easy, hedonistic lifestyle."[48]

Other documents submitted by B&W show that the company has attempted to capitalize upon the erroneous consumer perception that there is a health benefit to smoking mentholated cigarettes. Documents pertaining to the marketing of Kool cigarettes demonstrate that the company is aware of the consumer misperception about the relative safety of menthol cigarettes and utilizes it in the development of advertising strategies for Kools.[49]

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[47] These strategies were employed in a six-month media campaign conducted in three test cities in 1976. The advertising allotment for the campaign was approximately ten times the normal advertising dollar amount for a six month period. (Document A015486 - Memorandum from M.M. Matteson to V.C. Broach, July 14, 1976, emphases added).

[48] Document A080115 - "Viceroy Marketing/Advertising Strategy," January 26, 1976.

[49] In 1976, B&W held four focus group discussions to gauge menthol smokers' responses to a new Kool 120mm cigarette. The majority of the participants were menthol cigarette smokers. In a number of cases, the participants told B&W that they switched to menthol either for health considerations or from a general feeling that menthol cigarettes are less dangerous. According to B&W, a "pseudo-health image" has accrued to mentholated cigarettes. (Document A080675 - "Low Tar Longs Project - Creative Agency Assignment," 1977.) By characterizing the health image of mentholated cigarettes "pseudo," B&W admits its knowledge that menthol is of no health benefit to
(Continued)

A third set of documents obtained from Brown and Williamson reveal that in 1976, Brown and Williamson introduced a new brand of cigarette named Fact. The Brown and Williamson documents indicate that the company believed that Fact cigarettes were a new product which reduced the amount of harmful gas in the cigarette smoke inhaled by the consumer. Therefore, Fact was initially advertised as a brand with the unique ability to filter certain gases.[50] However, initial sales of Fact were not considered satisfactory by Brown and Williamson, so in 1977 it temporarily halted all advertising and promotion of the cigarette while it developed a new market strategy.[51]

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[49] (Footnote Continued)

smokers. In a 1978 document discussing Kool cigarettes' strengths and weaknesses, B&W also admits that one of Kool's strengths "rides on the connotation that menthol has health overtones." (Document A006981 - Memorandum from R.L. Johnson to P.E. McGowan, B&W, March 13, 1978.) In addition, B&W states that one of the strengths of its Kool Super Longs is that "menthol and 'tar' delivery has synergistic therapeutic implications." (*Id.*) B&W intends to exploit this false belief. In its document describing Kool's objectives through 1981, B&W states that its strategy will be to provide product safety reassurances while enhancing the satisfaction and refreshment perception. (Document A035669 - "Kool Three-Year Objectives," August 15, 1978.)

In fact, mentholated cigarettes tend to have a high "tar" and nicotine content.

[50] Document 7244 - "Fact 1976 Concept Description and Potential and Marketing Plan."

[51] Document 35523 - "Fact 1977 Repositioning and 1978 Marketing Plan Summary."

In April 1977, Brown and Williamson's advertising agency, Post-Keyes-Gardner, Inc., presented Brown and Williamson with its marketing and strategy recommendations for the reintroduction of Fact cigarettes.[52] The ad agency proposed two possible strategies to distinguish Fact from other cigarettes: 1) "More complete health protection through selective gas filtration," 2) "More taste and satisfaction in a low tar cigarette."[53] About the proposed strategy focusing on better health protection as the result of inhaling lower levels of gas, Post-Keyes-Gardner, Inc. wrote:

> A secondary opportunity to distinguish Fact from the mass of tar number claims is by capitalizing on the product's unique selective gas filtration. This would demand "product image" advertising and would provide the brand with a real point of difference. It would mean expanding the cigarette health issue beyond tar to encompass gas. However, this would require establishing "gas" as a meaningful health hazard in cigarettes because currently there is very low consumer awareness or comprehension of the gas problem. The Agency believes one of the major problems with the introductory advertising for Fact was that it failed to educate health concerned consumers about the dangers of gas. This failure to establish the gas problem meant that Fact's selective filtration promise was meaningless to the majority of the target audience. However, if smokers are effectively educated regarding this problem, the selective gas filtration promise may still be powerful, particularly among the very health conscious.[54]

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[52] Document 35524 - Brown & Williamson, "Marketing Advertising Strategy Recommendations for the Reintroduction of Fact Cigarettes," April 18, 1977. (The same document was also submitted by Post-Keyes & Gardner, Inc. - Document 714569).

[53] Id.

[54] Id., at 3.

However, the agency also noted the weakness of this proposed strategy:

> This strategic option assumes gas will become a major health issue. To ensure it becomes an issue will require an educational approach in introductory advertising. It is questionable whether any cigarette manufacturer should be publicizing a new health hazard for cigarette smokers. The desire to avoid spelling out the gas hazard in advertising could severely weaken the effectiveness of this approach.[55]

Ultimately, Brown and Williamson documents indicate that it elected not to educate the public about the health hazards associated with the gases in cigarette smoke and not to focus the Fact ad campaign on the low gas issue. The reason for Brown and Williamson's decision is explained in a document entitled "Fact 1977 Repositioning and 1978 Marketing Summary":

> Until the problem of gas becomes public knowledge through government investigation or media coverage, a low gas benefit will remain of little strategic value. [emphasis added][56]

The rationale is restated in a memorandum from the representative of the Brown and Williamson "Brand Group" which had overall responsibility for Fact:

> We do not support definition in advertising of the problem of gas in order to specifically communicate its consumer benefit and distinguish it from low "tar". To supply such definition would require overt references to the alleged ciliatoxic and cardiovascular ill effects of smoking. The possible ramifications of this in the Legal, Regulatory, and Policy areas are appalling...a likely result of such

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[55] *Id.*, at 6.

[56] Document 35523 - "Fact 1977 Repositioning and 1978 Marketing Plan Summary."

Statement by William J. Holayter
Legislative Director

International Association of Machinists
and Aerospace Workers

Mr. Chairman, Members of the Subcommittee:

As director of Legislation and Political Action for one of the nation's largest and most diversified unions I appreciate this opportunity to place our views on HR-5653 in the record. Briefly, this bill is designed to further discourage consumption of cigarettes not only in the United States but in every country to which American-made cigarettes are ~~expected~~ exported.

We include in our membership a number of skilled maintenance machinists in the tobacco industry. But lest you think we oppose HR-5653 on this ground, let me note that our far more sizeable membership in defense industries has not precluded our opposition to the further buildup of doomsday weaponry in the armed forces.

We are not opposed to government regulation where government regulation is needed and appropriate. We would, in fact, welcome even more regulation of safety and health hazards in America's work places.

We would welcome tighter government control of all the toxic substances being poured by polluting industries into the air we breathe and the water we drink.

We would welcome more government concern with the hazards of nuclear power and the perils inherent in the disposal of nuclear waste.

There are many kinds of hazards, risks, perils and dangers against which individuals cannot protect themselves. For dangers such as these government regulation is both appropriate and necessary. And a free people willingly accept the restrictions on individual liberty that such regulation imposes.

But a free society should not attempt to protect every citizen against every real or imagined danger. A free people must be free to make choices, to even take certain risks, so long as they do so with their eyes open.

By this time every adult in America has had ample opportunity to become aware of the dangers of smoking. To the drumbeat of continuous warnings over the past two decades, millions have voluntarily stopped smoking. Other millions have chosen not to start. That is their free choice. But it is respectfully submitted that if it were the only choice, it would be anything but free.

Government attempts to deny free choice to adults are self-defeating. Over the long run the prohibition of alcoholic beverages by the 18th Amendment did not decrease consumption of alcoholic beverages. In fact, it stimulated new sources of demand, especially among women and young people. Moreover, when government tries to coerce all adults into a mode of behavior dictated by the desires or prejudices of some, it becomes vulnerable to disrespect and ridicule or worse, as in the case of prohibition, corruption at every level. Government has no more right to try to stop all adults from smoking (because it is thought to be bad for health) than it has to try to make all adults eat yogurt (because it is thought to be good for health).

We recognize that HR-5653 was drafted for the best of motives. We acknowledge the sincerity of those who support such legislation. But we believe our opposition is based on equally good and sincere reasoning.

Freedom means the right to make reasonable choices. This principle seems more supportive of the personal liberties we cherish than the proposition inherent in HR-5653 — namely that government must wrap us all in a protective cocoon of regulation of personal habits to protect us against ourselves.

March 24, 1982

STATEMENT
of the
BAKERY, CONFECTIONERY & TOBACCO WORKERS INTERNATIONAL UNION
to the
HOUSE SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENT
Committee on Energy and Commerce

Re: H.R. 5653, "The Comprehensive Smoking Prevention Education Act of 1982"

The Bakery, Confectionery and Tobacco Workers International Union represents over 160,000 workers in the United States and Canada. Approximately 30,000 of our members are employed in the U.S. tobacco industry.

We present this testimony on behalf of all of our members - those who are employed in that industry, as well as those employed in the various aspects of the food production industry. We also speak on behalf of the many workers in the tobacco industry who are not organized and do not, therefore, have a collective voice to represent them at the hearings. We also represent many organized workers who are concerned with the impact of this bill, who were not provided time to testify, including machinists, electricians, carpenters, farm workers, distributive workers and retail workers, and others.

We oppose H.R. 5653, The Comprehensive Smoking Prevention Education Act, for two very important reasons.

First, it threatens our industry and our workers with the needless loss of sales, earnings, and ultimately, jobs.

Second, it threatens a host of other workers in other industries and the general public with the loss of protection against hazardous environmental and occupational exposures.

To put it plainly, this legislation is not what it seems to be. It is not merely a harmless labeling bill, but rather the first step down the road to prohibition. In fact, it is not merely a tobacco and health bill, but rather a red herring that could be used to divert attention from efforts to undermine other health policies and programs.

The stakes are very high and time for consideration of the full implication of this bill has been severely restricted.

Frankly, we have been concerned about the imbalance in the time allotted for testimony in support of this legislation versus the time for opposition views.

Three television performers were allowed to launch the hearings with personal statements in support of the bill, while only one labor union was permitted to testify, and then only as part of an industry panel which was itself denied the time it had requested for scientific witnesses. We believe it to be more important for the committee to hear the statements of those people whose livelihoods are directly affected by the proposed legislation.

The rush to judgment before all the facts are in will only do grave damage to the public interest and the credibility of the legislative process.

Mr. Chairman, this bill masquerades as labeling legislation, but there is an issue behind the issue. The hidden issue is whether national policy should shift from education to prevention, from choice to coercion.

This Committee will have to decide between those two roles for the government. Should the government continue to give people information so they can make their own free choice? Or should it aggressively persuade people to modify their behavior until they stop?

Present U.S. policy calls for the public to be informed, the measure of effectiveness being the extent of public awareness. Awareness stands at an astonishingly high level of 90 percent, verging on universal acceptance according to behavioral scientists.

The neo-prohibitionist strategy of this bill calls for prevention, and is based on the theory that if people reject the government's admonitions, they cannot really be informed -- and, therefore, must be reformed. The new measure of effectiveness shifts from knowledge to conformity.

Even on these terms, the present policy of education is working! The prevalence of smoking has dropped to 35 percent, the lowest ever recorded by the Gallup Poll. But apparently, it is not enough that awareness is at its highest level, and smoking, the disapproved behavior, is at its lowest point.

That more prevention is nevertheless prescribed betrays the neo-prohibitionist motivation that lies just under the surface.

This motivation explains why the bill loads packages and advertising with more warnings and lists than any other product is required to carry. It also explains why the bill opens the door to a massive overload of litigation. If these prohibitionist effects succeed in depressing sales by just one percent, the adverse impact would be significant. Based on data from a recent Wharton study of the tobacco industry's contribution to the U.S.

economy, we estimate that the loss for just nine of the states (California, Illinois, Kentucky, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Texas) would be more than 10,000 jobs and more than $170 million in wages. One-fifth of this loss would come from tobacco farming, manufacturing, retail sales, and suppliers; the remainder would result from the ripple effect on the rest of the economy.

Further economic hardship would result from the loss of export markets and of American jobs that depend on exports. This bill goes far beyond existing legislation by requiring warning labels on cigarettes produced in this country for export. Could American cigarettes carrying a health warning label compete with cigarettes which bear none? Can there be any doubt of the result on sales and on American jobs?

The American Cancer Society, a major lobbying force for this legislation, wants to cause more than a one percent drop in smoking. Several years ago, they launched their Target Five campaign, aimed at a 25% decrease in smoking in five years. And, it must be recognized that in Sweden, the source of this bill, the government's stated goal is to achieve a smoke-free nation by the year 2000.

We seriously question the wisdom of buying the Swedish import for Americans. We seriously question the wisdom of disrupting a health industry and creating more unemployment in a recession. We question the wisdom of setting up a new anti-smoking bureaucracy with unspecified spending authority when other essential health and social programs are being slashed. Mr. Chairman, we do more than "seriously question the wisdom" of this bill -- we reject its folly.

Now, let us turn to the second major ground for our opposition.

Section 3 deals with so-called findings. These blame every major chronic disease on smoking, and thereby create a smoke-screen for the occupational and environmental factors involved.

The very first one states that "the Congress finds that cigarette smoking is the largest preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually."

At first glance, it is difficult to conceive of a statement more alarming, more compelling, more demanding of remedial action. It calls for nothing short of outlawing tobacco.

But on reflection, this finding is curiously phrased; the words have an Alice-in-Wonderland quality. Their meaning is hard to pin down. For example, if cigarette smoking is the "largest preventable cause of illness," what are the second and third largest preventable causes? What are the "non-preventable" causes? Is smoking "preventable" while environmental pollution is not?

If these statements of findings had to be substantiated, as the FTC requires advertising statements to be, I doubt they would survive. The bill says flatly that smoking "is associated with" over 300,000 deaths a year. Yet, the first Surgeon General's Report in 1964 stated that:

> The total number of excess deaths causally related to cigarette smoking in the U.S. population cannot be accurately estimated.

The Committee which wrote the report considered the possibility of trying to make such calculations, but rejected the idea because "it involves making so many assumptions that the Committee felt that it should not attempt this...."

That restraint is as needed now as it was then.

We believe the findings in this bill are unsubstantiated and will be misused to the detriment of millions of workers exposed to occupational hazards. We oppose this bill and its findings to show our solidarity with:

- o Coal miners whose black lung disease has been blamed on smoking.
- o Textile workers whose brown lung disease has been blamed on smoking.
- o Asbestos workers whose lung diseases have been blamed on smoking.

And the list includes uranium workers, chemical workers, metal workers, shipyard workers, and many others. Public Health Service and the voluntary health organizations should honestly abandon the blame-the-victim approach and get at the truths of what is causing disease.

Earlier this year, this Committee heard testimony from two scientists who reported that "at least 11 percent and more likely 21 percent" of lung cancer in the U.S. can be attributed to air pollution. They noted that the proportion of adult smokers has decreased and that cigarettes now contain half the tar content of 20 years ago, yet lung cancer rates continue to climb. "To us this indicates that something else is at work," the scientists said.

But to the supporters of this bill, it's all cigarette smoking.

Recently, the National Wildlife Federation reported that "responsible scientists believe air pollution is responsible for about 50,000 excess deaths, seven million sick days, and 15 million days of restricted activity per year."

But to supporters of this bill, it's all cigarette smoking.

The lung cancer rate for white men along the coastal sections of Northern Florida, South Carolina and Georgia are among the highest in the nation. The National Cancer Institute says it may be the result of exposure in the booming shipbuilding industry during World War II. A Florida State University study attributes it to airborne chemical pollutants from industrial plants in New York and Illinois.

But to the supporters of this bill, it's all cigarette smoking.

The New York Times has recently looked at the growing controversy over whether environmental or lifestyle factors cause cancer. The former chief epidemiologist of the American Cancer Society, and other associated with the industrial establishment, believe that cancer-causing pollutants are relatively minor factors compared to factors such as smoking, diet, alcohol, and even sexual and reproductive behavior, a view that is consistent with the bill's "findings".

But the New York Times also reported that other scientists believe that factors other than smoking are involved; they are worried about "poisons escaping from smokestacks, toxic waste dumps, nuclear reactors." They are worried that "black men smoke less than whites, yet have higher lung cancer rates, perhaps because they have...more hazardous jobs."

But the supporters of the bill have no similar worries; to them, it's all due to smoking.

Politics makes strange bedfellows. H.R. 5653 has been introduced by a Member with a 100% AFL-CIO voting record; its Senate version has been introduced by a man with a zero AFL-CIO rating. We can't split the difference. We totally oppose both bills.

And in doing so, I would like to point to a report adopted by the AFL-CIO Executive Council in 1980:

> Some employers have exploited scientific studies of the combined effects of smoking with occupational exposure to toxic substances and conclude that it would be unnecessary to control exposure of these substances, if workers stopped smoking.
>
> The AFL-CIO is opposed to any coercive efforts to infringe on individual rights of individuals who smoke or of those who don't. We also oppose misuse of scientific data concerning smoking and exposure to toxic substances to serve as a rationale for failure to take necessary steps to prevent worker exposure to toxic substances in the workplace, which are shown to adversely affect their health.

We are impressed by the foresight of our Executive Council in stating two years ago the fundamental basis for rejecting this legislation today.

STATEMENT OF THE AMERICAN ADVERTISING FEDERATION ON THE ADVERTISING REQUIREMENTS OF H.R. 4957

The American Advertising Federation hereby submits its statement in opposition to H.R. 4957 and particularly to the requirement in Section 4(a)(2) that health warning disclosures must appear in all cigarette advertising.

The American Advertising Federation (AAF) is a national trade association which includes within its membership all of the various elements of the advertising industry. Its principal office is located at 1225 Connecticut Avenue, N.W., Washington, D.C. 20036. Its membership includes: newspaper publishers, magazine publishers, radio and television broadcasters and radio and television networks, advertising agencies and more than 400 companies which produce and/or advertise consumer products including cigarettes. Approximately 22 additional trade associations (including such diverse organizations as outdoor, transit, international, broadcasting, direct mail marketing, associations) with memberships comprised of companies engaged in various advertising pursuits are also members of AAF. The Federation membership also includes 209 local advertising clubs and federations located throughout the United States. These local organizations have a combined membership of approximately 25,000 advertising practitioners.

The interest and hence, the comments of the American Advertising Federation in this proceeding are limited to the advertising proposals contained in the bill. In particular we are concerned with the proposal to replace the current advertising "warning" with a rotating series of more specific warnings as described in Section 4(a).

In Section 2 the bill makes six "findings" as to the health consequences of smoking cigarettes and in Section 2(7) Congress "finds" that "present Federal, State, and private initiatives have been insufficient in conveying these health messages to the American public." According to Section 2(9), the bill constitutes "a new strategy . . . to educate and provide information to the American public to allow them to make informed decisions as to whether or not they should smoke."

Insofar as advertising is concerned the "new strategy" imposed by the bill is to require a rotating series of specific health warnings on labels and in advertising. Presumably the present warning on labels required by the Federal Cigarette Labeling and Advertising Act (15 U.S.C. 1333), the ban on broadcast advertising imposed by the same Act (15 U.S.C. 1335) and the FTC's orders mandating a warning in all print advertising are the "federal initiatives" which are now found to be "insufficient."

In introducing the bill Chairman Waxman referred to the conclusion of the FTC staff contained in its May 1981 Report on the Cigarette Advertising Investigation (File No. 792-3204) that "many consumers do not have enough information about the health risks of smoking . . ." But the same staff report acknowledges: "most people know that smoking is somewhat hazardous to health." Stf. Rept. 3-45. Apparently what the FTC staff and the bill's drafters feel is lacking is particular knowledge, in clinical detail, of the Surgeon General's findings as to the relationship between smoking and various diseases.

The essential fact is that people, probably all people, know, minimally, that "The Surgeon General has determined that cigarette smoking is dangerous to your health" and yet some of them continue to smoke. This bill is premised on the supposition that many smokers might quit if they possessed more precise information. But is that really true or only speculation? Moreover would a series of rotating advertising warnings implant that requisite knowledge?

Smoking and Health. The 1979 Report of the Surgeon General (hereinafter, the Report) sheds some light on these questions. In the first place advertising or the lack thereof appears to have little or no effect upon the incidence of cigarette smoking. In chapter 18 the Report cites studies which reveal that "bans on television advertising for cigarettes in several countries, including the United Kingdom, Denmark, Ireland, New Zealand, and Italy, seem to have had almost no effect on per capita cigarette consumption." p. 22. The Report points out that: "In communist countries, smoking is prevalent without advertising of any sort to support it." After citing statistics which show that per capita cigarette sales increased greatly during the period from 1922-1952 the report goes on to state:

> More recently, however, the cigarette market has been in a relatively mature, stable state and has had a much lower rate of growth. As the cigarette industry has asserted, the major action of cigarette advertising now seems to be to shift brand preferences, to alter market shares for a particular brand. p. 23.

If advertising plays little or no role in the implementation and continuation of the smoking habit then advertising can hardly be expected to be an effective medium for an attack on the habit. The Surgeon General's Report comes to the same conclusion. In chapter 17 the Report discusses a study, Television Advertising and the Adolescent, published by S. Ward in 1971. Ward concluded, according to the report, "The television medium appears to influence the formation of ideas and attitudes, yet does not 'trigger' adolescents to buy a product." According to the Ward study, teenagers listed cigarette advertisements as "least-liked." The Surgeon General's Report concludes:

> It is possible that because of cognitive and social differences in various development stages of children and adolescents, mass communications may not be the most appropriate means to reach children and adolescents with smoking-deterrent messages. Chap. 17, pp. 15-16.

The Surgeon General's Report specifically disagrees with the speculation that increased knowledge with respect to health hazards will affect smoking habits. At page 8 of chapter 17 the Surgeon General finds:

> As would be expected, beliefs of teenagers about smoking are related to whether or not they smoke. Of course, smokers generally hold more favorable attitudes toward smoking than do nonsmokers . . . Nevertheless, data . . . suggest that even teenage smokers seldom consider the decision to smoke a wise decision. For example, 77 percent of smokers believe that it is better not to start smoking than to have to quit. Over half of the teenage smokers believe that cigarette smoking becomes harmful after just one year of smoking. Eighty-four percent say it is habit-forming, while 68 percent agree that it is a bad habit. Of all teenagers, 78 percent believe that cigarette smoking can cause lung cancer and heart disease. Eighty-seven percent of all teenagers and 77 percent of the teenage smokers believe that smoking can harm their health.

Despite this knowledge, smoking among teenage boys remains constant and among teenage girls is actually increasing. Apparently the perceived social and psychological benefits of smoking outweigh the fear of adverse health consequences.

As the Surgeon General's Report points out, the reasons why people smoke are many and varied. Notably absent from the list is a lack of information as to health consequences. In the United States people smoke, obese people overeat, drivers refuse to fasten safety belts and motorcyclists refuse to wear helmets, not because of a lack of warning as to consequences, but simply because they refuse to heed such warnings. With this segment of the population additional warnings will serve no purpose and may indeed be counterproductive.

If this bill is enacted it will signal Congressional intention to transform the purpose and functioning of advertising in the United States for, in essence, it stands for the proposition that government can conscript private advertising to carry out national public education campaigns and force private advertisers to underwrite the costs of these campaigns. The bill leaps, from a finding that the public lacks precise information about the effects of cigarette smoking to a conclusion that cigarette advertising must be appropriated by government fiat to carry the government's message. This reasoning, if accepted, would create such a broad precedent as to sweep within its scope an innumerable array of advertised products. Moreover it ignores the Surgeon General's conclusions that advertising's major effect is merely to "shift brand preferences" and that "mass communications may not be the most appropriate means to reach children and adolescents with smoking-deterrent messages."

AAP believes that there has been no showing that advertising plays a role in the public's decision as to whether to smoke or abstain and that further usurpation of advertising space is unwarranted. We also believe that the proposed system will be no more effective than the present warning for neither are relevant to the underlying causes of behavioral smoking.

For these reasons we urge that H.R. 4957 not be enacted into law.

Dated: March 3, 1982

Respectfully submitted,

Howard H. Bell

Howard H. Bell
President

COMMENTS

of the

AMERICAN NEWSPAPER PUBLISHERS ASSOCIATION

The American Newspaper Publishers Association (ANPA) is a national trade association whose more than 1,400 member newspapers are responsible for publishing more than 90 percent of U.S. daily circulation. The Association includes a number of non-daily newspapers as well. We appreciate this opportunity to comment on H.R. 5653, The Comprehensive Smoking Prevention Education Act of 1982, legislation which we believe raises certain First Amendment issues which Congress should carefully consider.

Our concern centers on Section 4 of the bill. Section 4(a)(3) mandates that all cigarette advertising contain one of seven specifically-worded statements regarding the hazards of smoking. Section 4(b) directs the Federal Trade Commission to establish a system whereby the advertising for each brand of cigarettes shall include each of the seven warning statements over a seven-year period, with no statement to be carried for more than one year out of the seven. The section also requires that the warning statements be in a "conspicuous place" and "appear in conspicuous and legible type in contrast or background material . . . in the advertising."

While ANPA understands the public health concerns which have motivated this legislation, we believe the Congress should proceed with extreme sensitivity and great caution when it considers regulating speech as a method of regulating the sale and use of an entirely legal product.

By seeking to extend the warning label requirement to advertising, to specify the precise words to be used in seven different warning messages required to be contained in such advertising, and to direct the establishment of a new regulatory system by which the government would determine, in effect, when and by whom each warning message would be published, H.R. 5653 proposes a substantial expansion of governmental control over the commercial speech of advertisers and, indirectly, over the newspapers printing that speech. ANPA strongly suggests that the expansive regulatory scheme embodied in H.R. 5653 goes well beyond anything the record might support.

Advertising is the central means of communicating information required for economic decision-making in our free society. In a series of decisions including *Virginia State Board of Pharmacy v. Virginia Citizens Consumer Council, Inc.*, 425 U.S. 748 (1976), *Central Hudson Gas & Electric Corporation v. Public Service Commission of New York*, 447 U.S. 557 (1980), and most recently *In Re R.M.J.*, _____ U.S. _____ (1982), the U.S. Supreme Court has recognized that advertising enjoys significant First Amendment protections. While the Court has indicated that some of the full protection of political speech may not be accorded purely commercial speech, the burden is on the government to show a compelling need for regulation and also to show that the regulation is no more extensive than necessary. See *In Re R.M.J.*, *supra*. In classic First Amendment analysis, speech which is governmentally mandated is, if anything, even more suspect than speech which is restrained. See *Miami Herald Publishing Company v. Tornillo*, 418 U.S. 241 (1974).

Enactment of H.R. 5653 would be a significant escalation of regulation of speech in this area. It is one thing to mandate a warning label on cigarette packages and quite another to specify statutorily the precise language of seven specific massages which must be contained in advertisements as well as in packaging, and to provide for a system under which the questions of when those messages will be run and by which advertisers are determined by the government.

ANPA recognizes that the First Amendment protections offered commercial speech are not absolute, and that some forms of regulation are permissible. For example, place and manner restrictions are applicable, as is regulation of commercial speech that is false or deceptive. Similarly, there may be restrictions on advertisements for transactions or products which are illegal. See, e.g. Pittsburgh Press Company v. Human Relations Comm'n., 413 U.S. 376 (1973). None of those justifications for regulation of commercial speech applies to cigarette advertising. Although cigarette smoking may well be harmful to public health, it is not illegal, nor is cigarette advertising inherently misleading or deceptive. Likewise, the approach of H.R. 5653 is not a time, place or manner restriction in that it mandates publication of required content.

In Central Hudson Gas & Electric Corp. v. Public Service Commission of New York, 447 U.S. 557 (1980), the Supreme Court struck down a New York regulatory ban on promotional advertising by electric utilities. In his concurring opinion in that case, Justice Blackmun expressed concern about regulating speech that is not deceptive or misleading " . . . in order to manipulate a private economic decision that the state cannot or has not regulated or outlawed directly".

> The Court recognizes that we have never held that commercial speech may be suppressed in order to further the State's interest in discouraging purchases of the underlying product that is advertised. Permissible restraints on commercial speech have been limited to measures designed to protect consumers from fraudulent, misleading or coercive sales techniques.

447 U.S. at 574.

It may be argued that the warning messages required by H.R. 5653 are simply examples of the kinds of warnings and disclaimers that the Supreme Court, in several of the recent commercial speech cases such as *Virginia Pharmacy* and *In Re R.M.J.*, has indicated may be permissible forms of regulation. Uniformly, however, references to the possibility of requiring such disclaimers occur in the context of a discussion of commercial speech that is deceptive or misleading. For example, in *Virginia Pharmacy*, Justice Blackmun notes that

> The [attributes of commercial speech such as its greater objectivity and hardiness] may also make it appropriate to require that a commercial message appear in such a form, or include such additional information, warnings, and disclaimers, *as are necessary to prevent its being deceptive*.

425 U.S. at 771, n. 24 (Emphasis added).

There is no suggestion that cigarette advertising is so inherently deceptive or misleading as to require the mandatory inclusion of specifically-worded warning messages. This is not to say that cigarette advertising which is deceptive or misleading is immune from challenge. Under Section 5 of the Federal Trade Commission Act, (15 U.S.C. Sec. 45), the FTC clearly has the power to deal with such advertising. Absent such a specific finding, it should not be assumed that cigarette advertising per se is deceptive without a health warning, any more than liquor advertising would be deceptive without a warning about the dangers of alcoholism.

In the *Central Hudson* case decided in 1980, the Supreme Court set out a four-step analytical framework for examining restrictions on commercial speech:

> In commercial speech cases, then, a four-part analysis has developed. At the outset, we must determine whether the expression is protected by the First Amendment. For commercial speech to come within that provision, it at least must concern lawful activity and not be misleading. Next, we ask whether the asserted governmental interest is substantial. If both inquiries yield positive answers, we must determine whether the regulation directly advances the governmental interest asserted, and whether it is not more extensive than is necessary to serve that interest.

447 U.S. at 566.

When this four-part test is applied to H.R. 5653, the first two inquiries can be answered positively: cigarette advertising, in general, is not misleading and is therefore protected by the First Amendment, and the asserted governmental interest — public health — is substantial. It is in connection with the last two questions that ANPA believes the commercial speech provisions of H.R. 5653 do not satisfy this constitutional test: whether mandatory warning messages in cigarette advertising "directly advance[s] the governmental interest asserted," and more importantly, whether the government's intrusion into commercial speech "is not more extensive than is necessary."

Whether a series of seven warning messages carried in cigarette advertising "directly advances" the governmental interest in public health is open to serious question and should at least be the subject of careful debate and specific factual showings. There is already a required health disclaimer in cigarette advertising and packaging; if that has been ineffective, what evidence is there that the new multiple warnings will effectively advance the government's interest?

A more critical inquiry is whether the kind of approach represented by H.R. 5653 unnecessarily involves the government in a pervasive system of regulating non-deceptive commercial speech. Is it essential, for example, that the FTC be given the power to dictate which advertising will carry which message and for how long?

No matter how seemingly laudable the goal, any legislation which proposes to delegate to government the power to decide what will be published and when something will be published is constitutionally most troublesome. Equally troubling is the proposed government requirement that specific words must be published in not just one but seven different messages. ANPA believes this would impinge on the constitutionally-protected domain of advertisers and the press.

Commercial speech restrictions must be a "last resort" action of government. Only where there is a concrete showing that an identifiable problem cannot be solved by means other than restricting speech can the government turn to measures such as those embodied in this legislation. The process of reaching this decision must include a complete assessment of all the information now available to the public concerning cigarettes and the health issues surrounding them. Having taken that and all other factors into account, then, and only then, can restrictions on commercial speech be considered. It is not at all clear that this rigorous process has been followed in advancing these legislative measures.

The principal concern of ANPA about this legislation is the proposed resort to government regulation of speech as a means of regulating a product which for whatever reason, the government chooses not to regulate directly.

We believe a very troubling precedent is set whenever the federal government determines the content of the speech of private entities. H.R. 5653 not only proposes to do that, but also would give a regulatory agency the power to determine when certain messages would be published, for how long and by whom. Absent compelling circumstances and clear evidence that other, less intrusive, alternatives are ineffective, this kind of governmental action is inimical to the preservation of free speech and a free press in our free society; thus the case has not been articulated which would support as harsh a statutory restriction on commercial speech as that embodied in H.R. 5653.

PUBLIC VERSION

FEDERAL TRADE COMMISSION

STAFF REPORT ON THE CIGARETTE ADVERTISING INVESTIGATION

BY:

Matthew L. Myers
Program Advisor

Craig Iscoe
Carol Jennings
William Lenox
Eleanor Minsky
Andrew Sacks

APPROVED:

Collot Guerard
Deputy Assistant Director
Division of Advertising Practices

Wallace S. Snyder
Assistant Director
Division of Advertising Practices

CONCUR:

James H. Sneed
Director
Bureau of Consumer Protection

MAY, 1981

In addition to the information in this Report, the Commission has also relied upon confidential information provided by the cigarette companies and their advertisement agencies pursuant to subpoena which has been deleted from the Introduction and Chapters II, III, IV and V of the version of the report released to the public. The beginning of each of these chapters describes the confidential material deleted.

INTRODUCTION*

In 1964, following the issuance of the Report on Smoking and Health of the Advisory Committee to the Surgeon General,[1] the Federal Trade Commission found that the mounting evidence of the "grave hazards to life and health" caused by cigarette smoking, together with the failure of the cigarette manufacturers to warn consumers of this danger, constituted "an unfair or deceptive act or practice within the meaning of Section 5 of the Federal Trade Commission Act."[2] The Commission concluded that if consumers did not know about the health risks of smoking, cigarette advertising which failed to disclose these dangers was unfair or deceptive. Prompted in 1969 and in 1971 by the same concern, the Commission again took formal action to require cigarette manufacturers to better inform the public in their advertisements about the dangers of smoking. As a result, all cigarette advertisements today contain the same health warning Congress required on cigarette packages, "Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous To Your Health."[3]

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* In addition to the information in this report, the Commission relied on confidential information submitted by the cigarette companies pursuant to subpoena. The confidential information has been deleted from the version released to the public. The locations where the information has been deleted are noted [Confidential Information Omitted].

[1] U.S. Department of Health, Education and Welfare, Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service (1964) ("1964 Surgeon General's Report").

[2] The Trade Regulation Rule for the Prevention of Unfair or Deceptive Advertising and Labeling of Cigarettes in Relation to the Health Hazards of Smoking, 29 Fed. Reg. 8324, 8325, 8356 (July 2, 1964). ("The 1964 Cigarette Rule"). For a detailed discussion of previous actions of the Federal Trade Commission with regard to cigarette advertising, see Chapter IV, Section I, infra.

The need to inform consumers adequately about the serious health consequences of cigarette smoking has also been a major concern of Congress. In 1965 Congress passed the Federal Cigarette Labeling and Advertising Act requiring all cigarette packages sold or distributed in the United States to contain the following warning: "Caution: Cigarette Smoking May Be Hazardous to Your Health." In addition, the Act required the Federal Trade Commission to transmit a report to Congress annually on the effectiveness of cigarette labeling, current cigarette advertising practices and the need for additional legislation.[4] In 1970 Congress passed the "Public Health Cigarette Smoking Act" requiring all cigarette packages to contain the current revised warning and making it unlawful to advertise cigarettes on radio or television.[5]

As in 1964, this is a particularly appropriate time for consideration of whether current cigarette advertising adequately warns consumers of the health hazards attributable to smoking and whether new action is needed. In 1979 the Surgeon General issued the most comprehensive report on smoking and health in 15 years.[6] The 1100 page 1979 Report, based on an analysis of more than 30,000 articles and scientific studies on the relationship between tobacco consumption and health, confirms and expands the conclusions of the 1964 Surgeon General's Report. The 1979 Report confirms the evidence demonstrating the relationship of smoking to diseases, such as cancer, heart attack and chronic obstructive lung disease. It also establishes the relationship between smoking and a number of other health hazards about which little was known in 1964. These include the effects of smoking during pregnancy on the developing fetus and the newborn child, the increased risk of lung cancer in women who smoke, the interaction between smoking and birth control pills and the interaction between smoking and certain occupational hazards.

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[3] Lorillard, et. al., 80 F.T.C. 455 (1972).

[4] 15 U.S.C. §1331 et. seq (1965).

[5] 15 U.S.C. §1331 et. seq. (1970).

In November 1980, the Surgeon General issued another major report entitled, Promoting Health/Preventing Disease: Objectives For The Nation.[7] This report sets out specific objectives for fifteen priority areas designated by the Surgeon General as the most essential for achieving our national health aspirations and goals. Smoking is one of the fifteen priority areas designated. In fact, this Report finds that smoking is this nation's "single most important preventable cause of death" and that particular attention needs to be given to groups the Surgeon General designates as "high risk groups," such as "pregnant women, children and adolescents who initiate smoking at a young age."[8] Indeed, more teenagers are smoking today than ever and they are starting to smoke at a younger age. The Surgeon General concludes that more can and must be done to inform the public about the health threat caused by smoking. He then makes a number of specific recommendations, including:

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[6] Office of Smoking and Health, U.S. Department of Health, Education and Welfare, Smoking and Health: A Report of the Surgeon General (1979) ("1979 Surgeon General's Report").

[7] Office of Smoking and Health, U.S. Department of Health and Human Services, Promoting Health/Preventing Disease: Objectives For The Nation (1980).

-continuing the FTC requirement of a health warning in advertising;

-increasing the visibility and strengthening the content of the present warning to give the consumer additional needed information on the specific multiple health hazards of smoking, giving special consideration to rotational warnings and to the identification of special vulnerable groups;

-conducting educational campaigns directed both to the general public and to specific groups, such as women, especially pregnant women, youths, workers exposed to toxic substances and to others at special risk.[9]

The American Medical Association also has long been concerned about the need to inform consumers about the health hazards of smoking. In 1953, it banned tobacco advertising from AMA publications.[10] In 1968 the AMA House of Delegates adopted a resolution calling for the AMA to take a strong stand against smoking with every means at its disposal.[11] Recently, the AMA Council on Scientific Affairs made several recommendations regarding smoking, including a recommendation that the AMA urge physicians to alert their patients to the risks of smoking and encourage them to quit smoking. The Council also urged the AMA to recommend that the FTC seek to eliminate the use of role models in cigarette advertising and that it require warning labels to be displayed on packages, advertisements and billboards.[12] In addition, in recent years several members of the life insurance industry have publicized the fact that they offer preferred rates to nonsmokers because they have concluded that nonsmokers are better health risks than smokers.[13]

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[8] Id. at 117.

[9] Id. at 119-123.

In December 1980, the President's Advisory Committee For Women issued its report, entitled *Voices For Women*,[14] on the status of women in the United States. While this is the ninth Presidential Advisory Committee report on the subject, it is the first such report to recognize smoking as an issue of particular significance to women.[15] As the President's Advisory Committee notes, although for many years smoking has been seen as a man's health problem, "[s]moking may well prove to be the major health problem facing women in the 1980's."[16] The Advisory Committee also finds that smoking is the "leading controllable cause of rising morbidity and mortality in adult American women,"[17] and that the Office of Smoking and Health of the Department of Health and Human Services, the federal agency with primary responsibility for public education programs on smoking, is "understaffed, underhoused and underfunded."[18] As did the Surgeon General, the President's Advisory Committee concluded that more must be done to increase public awareness of the health hazards of smoking.

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[10] Smoking and Health Council on Scientific Affairs, 243 Journal of the American Medical Association 779 (1980).

[11] *Id.*

[12] *Id.*, at 781.

[13] See, e.g., Cowell, M.J. & Hirst, B.L., State Mutual Life Insurance Company of America, Mortality Differences Between Smokers and Nonsmokers (1979).

[14] 1980 Report of the President's Advisory Committee for Women, *Voices For Women* (1980).

The findings and recommendations of these reports and the fact that it has been nearly a decade since the Commission and Congress last reviewed whether consumers were being adequately warned about the health effects of smoking indicate the need for a re-examination of this issue. This particular investigation was begun in May 1976 to determine whether Congress and the Commission's prior actions with regard to cigarette advertising, or the independent action of cigarette manufacturers in the interim, had effectively remedied the deceptive practices which previously had caused Congress and the Commission to act.

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[15] Id. at 50.

[16] Id. at 50, 57.

[17] Id. at 58.

[18] Id.

The Commission staff focused its present investigation on five major issues. They are: (1) In light of the substantial growth of medical information in recent years, what are the known health consequences of smoking? (2) What health information does cigarette advertising contain about the nature, probability and severity of the dangers of smoking? (3) Does the public already know these facts, or does a substantial portion of the population lack knowledge of or hold false beliefs about the dangers of smoking? (4) Is the current warning effective in alerting the public about the health hazards of smoking? (5) Is remedial action necessary? If so, what remedial measures, if any, are likely to result in the public being provided with sufficient health information to avoid any possible deception in cigarette advertising?

The issues presented by the current investigation are more complex than they were when the Commission first took action in 1964. In 1964, the Surgeon General had just published his first comprehensive report on smoking and health, and cigarette packages and advertisements contained no health warnings. Similarly, in 1969 and 1971, cigarette advertisements contained no health warnings. Thus, while the focus has previously been on the threshold question of whether to require a health warning at all, it now is necessary for the first time to examine the effectiveness of the warning -- the remedy adopted a decade ago -- and determine whether current cigarette advertising may be deceptive.

This report discusses in detail the results of the staff's investigation. As part of its investigation, staff has conducted an extensive review of the medical literature, including the Surgeon General's reports on smoking and the Tobacco Institute's response. It has sought out and examined the literature which discusses the views and findings of those who disagree with the Surgeon General's conclusions and has consulted with many of the foremost medical experts in the field. To evaluate the extent of consumers' knowledge of the health risks of smoking, staff conducted an exhaustive review of the available survey data and sponsored two additional nationally projectable surveys. The staff's evaluation of the current warning is based on the results of several additional studies, the analysis of the consumer knowledge data and the advice of communications and advertising experts, including a New York advertising agency. Similarly, to evaluate the remedial options, the staff conducted several studies and worked closely with both distinguished advertising experts and the same New York advertising agency.

SUMMARY OF FINDINGS [19]

The past efforts of the Commission plus the efforts of Congress and other governmental agencies and private organizations to increase the amount of health information available to consumers have had an important impact. Many more consumers now are aware that smoking is hazardous to their health than in 1964. The percentage of Americans who smoke cigarettes has declined significantly over the same time period, and a substantially larger number of those who do smoke now smoke cigarettes with lower levels of "tar" and nicotine.

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[19] The facts upon which this summary is based and the supporting data and sources for these facts are contained in the remaining Chapters of this report.

Nonetheless, the problems which prompted the Commission to act in the past still exist. While most Americans are generally aware that smoking is hazardous, some consumers, especially smokers, do not know this basic fact. However, even if it is assumed that every consumer is aware that smoking is hazardous, the evidence indicates that many consumers do not have enough information about the health risks of smoking in order to know how dangerous smoking is, i.e., what is the nature and extent of the health risk of smoking. Many consumers also do not know whether the general health risks of smoking have any personal relevance to themselves or whether they are among those groups of people who may be uniquely vulnerable to these health hazards. Finally, without more specific, concrete information, consumers have a more difficult time remembering and are less likely to consider health information at all in making their smoking decision.

More specifically, the data discussed in Chapter III suggest that many consumers do not know enough about the health effects of smoking to know how dangerous smoking is and indeed desire more information about the specific hazards of smoking. For example, the data indicate that many do not know what diseases are smoking related. Over 30% of the public is unaware of the relationship between smoking and heart disease. Nearly 50% of all women do not know that smoking during pregnancy increases the risk of stillbirth and miscarriage. Approximately 30% of those polled do not know about the relationship between smoking, birth control pills and the risk of heart attack.

Some of the health consequences of smoking, such as lung cancer and emphysema, are more well known. However, even for lung cancer, the most well known health effect, some gaps in consumer knowledge are evident. Thus, approximately 20% of those polled do not know that smoking causes cancer.

The data also indicate that substantial numbers of consumers seriously misunderstand and underestimate the increased risk of suffering these health problems as the result of smoking. For example, the survey data discussed in Chapter III indicate that over 40% of those polled did not know that smoking caused most (80%) cases of lung cancer and nearly one-quarter of those polled did not even know it causes many cases. Similarly, although the evidence indicates that smoking causes about 70% of all cases of chronic obstructive lung disease (emphysema and chronic bronchitis), approximately 60% of the public does not know that most cases of emphysema are smoking-related, and one-third of those polled are even unaware that many cases of emphysema are smoking-related. In addition, over 50% of the population does not know that smoking causes many as opposed to just a few cases of heart attack.

As the evidence in the section in Chapter III on "Overall Risk" demonstrates, many more consumers seriously underestimate the severity or increased risk of dying from these smoking-related illnesses. A thirty year old smoker will shorten his life on the average by between six to eight years if he smokes a pack or more a day. However, among those polled in the 1980 Roper study, 30% of the population and 41% of the smokers did not know that a typical thirty-year old male shortened his life expectancy at all by smoking.

Finally, the survey data indicate that a large number of people do not believe that they will personally suffer the health consequences of smoking. Thus, although the medical evidence clearly indicates that smoking only a few cigarettes a day can be harmful, nearly one-third of those polled and 40% of smokers polled believed that only heavy smoking is dangerous. Similarly, although the Surgeon General has concluded that low "tar" cigarettes have not been shown to be safe, over one-third of the smokers polled falsely believe that it has been proven that by smoking low "tar" cigarettes, smokers do not significantly increase their health risks over nonsmokers. Another example may be particularly important for nonsmokers, especially young people deciding whether to experiment with smoking. Although many experts now classify cigarettes as an addictive substance, many teenagers believe smoking is okay if they quit before it becomes a habit and approximately 50% of all those polled did not know that smoking may be addictive.

The importance of the fact that many consumers do not know about the health effects of smoking is heightened by the following. The medical evidence gathered over the past two decades indicates that cigarette smoking is far more dangerous to health than was thought in 1964. Smoking causes more than 300,000 deaths annually (one out of seven of all deaths) in this country. Last year alone over 80,000 people died from lung cancer caused by smoking. An additional 34,000 died from smoking-related emphysema and chronic bronchitis and well over 200,000 heart attack deaths were attributable to smoking. Recent research has strengthened the evidence demonstrating the relationship of smoking to lung cancer and other cancers, heart attack and chronic obstructive lung disease (emphysema and bronchitis). It has also established the relationship between smoking and a number of other health hazards about which little was known in 1964. These include the effects of smoking during pregnancy on the developing fetus and the newborn child, the development of lung cancer in women and the interaction between smoking and birth control pills.

In 1978, 54 million Americans smoked a total of 615 billion cigarettes. Many of those smokers are uninformed about the serious health consequences of smoking. The gap between the documented health effects of smoking and consumers' knowledge of these dangers is illustrated by a comparison of the following statements summarizing the health consequences of smoking (Summary A) with the following chart summarizing the best available survey data of the public's knowledge in this area (Summary B):

Summary A

HEALTH CONSEQUENCES OF SMOKING[20]

OVERALL RISK

-Cigarette smoking is the greatest environmental cause of death in the U.S.; well over 300,000 deaths annually are smoking related.

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[20] For a discussion of the evidence from which this chart is compiled, see Chapter I, Section II.

-To put this in perspective, annually more than one out of every seven deaths in this country are smoking related.[21] Each year six times as many people in this country die from smoking related causes as die from automobile accidents.[22]

-Overall, a smoker is 70% more likely to die at a given age than is a comparable nonsmoker.

-A two pack or more a day smoker decreases his life expectancy more than eight years; one pack or more a day, six years.

-The earlier one starts smoking, the more likely one is to die from it.

CANCER

-Smoking is the number one cause of lung cancer in this country, accounting for 80% of all lung cancer deaths.

-Last year, more than 80,000 people died of lung cancer caused by smoking.

-70% of lung cancer patients die within one year, 90% within five years.

-Smokers are 10 times more likely to die from lung cancer than nonsmokers. Heavy smokers (two packs or more per day) are 20 times more likely.

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[21] For example, the total number of deaths from all causes in this country in 1978 was 1,931,100. American Heart Association, Heart Facts 1978 at 9.

[22] National Highway Traffic Safety Administration statistics indicate that 50,226 people were killed in the U.S. in 1978 in automobile accidents. National Highway Traffic Safety Administration, U.S. Department of Transportation, 1978 Annual Report: Fatal Accident Reporting System, September 1979.

OTHER CANCERS

-Cigarette smoking has been established as a significant cause of cancer of the larynx, oral cavity, esophagus, and bladder, and is significantly associated with cancer of the pancreas and kidney.

-There is a synergistic relationship between smoking and use of alcohol that greatly increases the risk of cancer of the larynx, oral cavity, and esophagus for those who smoke and drink heavily.

-Estimated 1980 cancer deaths (other than lung cancer deaths) attributable to smoking:

Types Of Cancer	Total Deaths X Percent Attributable to Smoking	Total Deaths Attributable to Smoking
Larynx	3500 X 65%	2275
Esophagus	7600 X 60%	4560
Oral	8800 X 56%	4928
Bladder	10300 X 30%	3090
Renal	7900 X 30%	2370
Pancreas	20900 X 30%	6270
		23,493

Thus, approximately 40% of all cancer deaths in these categories is smoking-related.

HEART DISEASE

-Heart disease accounts for nearly one-half of all deaths in this country; cigarette smoking accounts for 1/3 of all heart disease deaths.

-In 1978, 225,000 heart disease deaths were attributable to cigarette smoking.

-Smoking is one of three major risk factors of heart attack.

-Smoking nearly doubles a person's risk of heart attack.

CHRONIC OBSTRUCTIVE LUNG DISEASE (Emphysema and Chronic Bronchitis)

- Smoking is the major cause of both emphysema and chronic bronchitis.
- 70-80% of all emphysema and chronic bronchitis deaths each year are attributable to smoking.
- In 1979, there were more than 34,000 smoking-related emphysema and chronic bronchitis deaths.

PREGNANCY AND INFANT HEALTH

- Maternal smoking during pregnancy significantly increases the risk of spontaneous abortion.
- Maternal smoking during pregnancy increases the risk of still-birth or infant death within a month of birth by at least 20% for light smokers (less than one pack a day), and by 35% for those who smoke more than one pack a day.
- Mothers who smoke have been found to be 36%-47% more likely to give birth prematurely.
- Babies born to women who smoke during pregnancy weigh an average of 200 grams (about half a pound) less than babies born to comparable women who do not smoke during pregnancy.

BIRTH CONTROL PILLS

- A woman who smokes and takes birth control pills is 10 times as likely to suffer a heart attack as a woman who does neither.
- A woman who smokes and takes birth control pills is 20 times more likely to suffer stroke by cerebral hemorrhage than a woman who does neither.

CARBON MONOXIDE

-Carbon monoxide is one of the harmful ingredients of cigarette smoke.

-Carbon monoxide reduces the amount of oxygen delivered to the cells.

-Carbon monoxide in the levels found in cigarette smoke is dangerous to people with heart or lung disease.

-Carbon monoxide is suspected to be the most important factor in causing spontaneous abortion, stillbirth, neonatal death, reduced birth weight babies and other complications associated with maternal smoking during pregnancy.

-Carbon monoxide may be responsible for causing increased risk of heart disease associated with smoking.

The overall extent to which millions of consumers lack knowledge of or hold false beliefs about these basic, important medical facts is in part revealed by the following chart:

SUMMARY B
KNOWLEDGE CHART[a]

AREA	Medical Fact Study Tested	% of total population unaware[b]	% of smokers unaware[c]	# of Adults unaware[d]	# of smokers unaware[e]
OVERALL RISK	Smoking is hazardous to health - Gallup, 1978 Roper	9-10%	13-17%	15 million	9-10 millic
	A thirty-year old man reduces his life expectancy by smoking a pack a day - 1980 Roper	30%	41%	50 million	20 million
	Light smoking is dangerous - 1978 Roper	31%	40%	50 million	20 million
CANCER	[Confidential Information Omitted]				
	Smoking causes lung cancer - Gallup,	19-23%	27-38%	30- million	15-million
	[Confidential Information Omitted]				
	Smoking causes most cases of lung cancer - 1980 Roper	43%	49%	70 million	25 million
	Smoking causes cancer of the mouth and esophagus - Gallup, Chilton[f]	15-21%	15-27%	25-35 million	10-15 millic

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a/ For a description of the surveys upon which this chart is based, see Ch. III.

b/ Percent unaware includes those who say they do not believe true statements, believe false statements, underestimate on a multiple choice question, or answer "don't know" or "uncertain". When there are two or more studies, the results are presented as a range.

c/ Computed as described in Fn. b, *supra*.

d/ Based on 160 million adults 17 years of age and older. See, Surgeon General's 1979 Report, Appendix A-11.

e/ Based on 54 million smokers. See, Surgeon General's 1979 Report, A-11.

f/ All references to Chilton in this study are based on the adult sample. Chilton also tested teens, and gave separate results for their teen sample.

AREA	Medical Fact Study Tested	% of total population unaware	% of smokers unaware	# of Adults unaware	# of smokers unaware
	Smoking is a major cause of cancer of the mouth, esophagus, and larynx - 1980 Roper	32%	39%	50 million	20 million
HEART DISEASE	Smoking causes heart disease - Gallup,	32-	37-	50- million	20- millic
		[Confidential Information Omitted]			
	Smoking is a major cause of heart disease - Chilton	40%	45%	60 million	25 million
	Smoking greatly increases risk of heart attack - 1980 Roper	25%	31%	40 million	15 million
BRONCHO-PULMONARY DISEASE	Smoking causes bronchitis and emphysema- Chilton,	9-	9-	15- million	5- millio
		[Confidential Information Omitted]			
	Smoking causes many cases of bronchitis and emphysema - 1980 Roper	34-55%	39-60%	55-60 million	20-30 milli
	Smoking causes most cases of bronchitis and emphysema - 1980 Roper	59-82%	63-85%	95-130 million	35-45 milli
PREGNANCY AND INFANT HEALTH	Maternal smoking increases the risk of miscarriage and stillbirth - Chilton, 1980 Roper	47[9]-40%	54-58%	75 million	30 million
ADDICTION	Smoking is addictive - 1978 Roper	54%	49%	85 million	25 million

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[9] Based on women in the 1980 Roper study.

AREA	Medical Fact Study Tested	% of total population unaware[b]	% of smokers unaware[c]	# of Adults unaware[d]	# of smokers unaware[e]
LOW TAR/ NICOTINE	It has not been proven that low-tar and nicotine cigarettes do not significantly increase risk of disease. 1980 Roper	33%[h]	36%[h]	55 million	20 million
CARBON MONOXIDE	Cigarette smoke contains carbon monoxide - Chilton, 1980 Roper	45-53%	38-53%	70-85 million	20-30 millio

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h/ Not including those who answered "Don't know if it's true."

As a result of these findings, staff undertook a re-evaluation of the current warning. After careful examination, staff believes that the current warning is not effective. The number of persons who understand that smoking is a health problem has increased in the last decade. However, surveys show that the public still lacks enough information about the risks of smoking to appreciate how dangerous it is. This gap in knowledge may be due to the fact that the current warning refers to the health hazard of smoking only in the most generalized way. The warning does not communicate information on significant, specific risks that have recently been identified. Furthermore, after nearly a decade the current warning has been overexposed and has simply worn out. This conclusion is further supported by data indicating that less than 3% of adults exposed to cigarette ads ever even read the warning. While cigarette ads present their message in a variety of frequently changing, attention getting formats using numerous image provoking, personalizable themes, the current abstract warning in the same rectangular shape has appeared unchanged in every cigarette ad for so long that few people ever notice or pay attention to it. The failure of the warning to provide specific health information and the abstract nature of the current warning, which makes it more difficult for consumers to relate the warning to themselves, contribute to its ineffectiveness. Thus, while the current health warning may have represented a reasonable remedial effort nearly a decade ago based upon the evidence then available, the continued growth of medical knowledge about the health consequences of smoking and new evidence about the present ineffectiveness of the current warning indicate that a new informational remedy may now be necessary.

SUMMARY OF RECOMMENDATIONS

Based upon the evidence obtained in this investigation, staff is concerned that current cigarette advertising practices may mislead consumers by omitting material facts about the health risks of smoking. Staff has also tentatively concluded that additional action designed to provide consumers with more information about the health consequences of smoking is necessary. In Chapter V the staff has considered a number of remedial options, including: (a) educational efforts of other governmental and private organizations; (b) voluntary industry self-regulation; (c) alteration of the size and shape of the warning; (d) replacement of the current warning with a single new, more specific warning; (e) replacement of the current warning with a rotational warning system; (f) placement of limitations on the use of imagery in cigarette advertising, known as "tombstone advertising;" and (g) disclosure of carbon monoxide levels.

Of the options explored, staff at this preliminary stage believes the following are likely to be most effective: (a) additional funding for expanded educational efforts, such as public service announcements; (b) changing the shape and increasing the size of the current warning; and (c) replacing the current warning with a system of short rotational warnings.

Expanded educational efforts, such as public service announcements broadcast during prime viewing hours, would reach millions of consumers. Changing the size and shape of the warning would improve its noticeability, but would not provide consumers with the additional necessary health information. Replacing the current warning with a more specific, single new warning would be an improvement; however, having more than a single warning would allow greater information to be available to the public and, thereby, decrease the possibility of deception. Rotating the various health warnings would also assist in maintaining their noticeability over an extended period, and would more effectively communicate a substantial amount of specific health information about which millions of consumers are uninformed. To be more effective these changes should involve the warning both on cigarette packages and in cigarette advertisements. While the adoption of any one of these remedial options by itself would not eliminate the problems discussed in this report, the adoption of the three options staff tentatively believes to be most effective as part of an overall educational effort would provide the public with additional health information and remedy any possible deception in cigarette advertising.

In light of the findings, conclusions and recommendations in this report, the staff recommends that: (a) copies of the staff report should be provided to Congress for its consideration, (b) the report should be released for public comment, (c) the Commission should continue its investigation, while working with Congress, members of the industry and appropriate governmental and private organizations to coordinate and determine what action should be taken by whom; and (d) after the close of the comment period, the staff should report back to the Commission with an analysis of the information obtained from public comments and a further recommendation as to whether additional or formal Commission action is necessary and appropriate.

CHAPTER I. THE PREVALENCE AND HEALTH EFFECTS OF SMOKING

I. SMOKING PREVALENCE

In order to evaluate the significance of the limited health information available from cigarette advertisements and the fact that many consumers do not know about many of the most significant facts about the health effects of smoking (See Chapters II and III, infra), it is necessary to examine the evidence on smoking trends and the prevalence of cigarette smoking in this country. Since the issuance of the Surgeon General's Report in 1964, the percentage of Americans who smoke cigarettes has declined from 42% of the population in 1964 to 33% of the population in 1978.[1] During this time period, health warnings were placed on cigarette packs and ads, cigarette advertising was banned from the broadcast media, several Surgeon General's reports on the hazards of smoking were issued, and a number of education efforts designed to increase the public's knowledge of the health hazards of smoking were undertaken.[2]

As the percentage of smokers in the population has changed, so too has the cigarette that is being smoked. There have been two significant developments in cigarettes over the past 30 years. The first was the replacement of regular cigarettes with filtered ones. Filter-tipped cigarettes, which were introduced in 1952, accounted for 65% of consumption in 1964 and comprise over 90% of the market today.[3] The second change has been the reduction in "tar" content of cigarettes sold. Cigarettes with less than 15 mg. of "tar" have increased their market share from 2.0% in 1967 to 40.9% in 1979.[4]

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[1] Office of Smoking and Health, U.S. Department of Health, Education and Welfare, Smoking and Health: A Report of the Surgeon General (1979) ("1979 Surgeon General's Report"), Appendix at 11.

[2] A recent study by the Commission's Bureau of Economics found that while it is not possible to determine the precise impact of each of these individual informational efforts on the decline in cigarette consumption, these educational programs did have a beneficial impact on consumer behavior as well as on consumer knowledge. Bureau of Economics, Staff Report on Consumer Responses to Cigarette Health Information, August 1979.

In 1978, over 54 million smokers smoked 615 billion cigarettes.[5] In 1979, total U.S. consumption of cigarettes rose to 620 billion.[6] In the first six months of 1980, Americans smoked approximately 315 billion cigarettes, an increase over the same period of 1979.[7]

Smoking rates have declined significantly only among adult males, and have increased among teenaged women. The percentage of adult male smokers went from 51.1% in 1965 to 37.5% in 1978.[8] The decline has not been as large with women smokers. Between 1964 and 1979, the percentage of adult women smokers declined by 5%, from 33%

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[3] Id.

[4] Most of these sales are of cigarettes with more than 9 mg. but less than 15 mg. "tar." Less than 11% of sales in 1979 were for cigarettes with 9.0 mg. "tar" or below. Preliminary figures for the F.T.C.'s Annual Report to Congress on Cigarette Advertising for the Year 1979.

[5] 1979 Surgeon General's Report, Preface at vii.

[6] U.S. Department of Agriculture, Tobacco Situation, September 1980 at 5.

[7] Id.

[8] 1979 Surgeon General's Report, Appendix at 9.

to 29%.[9] Most of this decline has taken place since 1976,[10] but, in 1978, almost 25 million adult women smoked.[11]

Smoking prevalence among young people has also not declined as significantly as among adult males. Between 1968 and 1979 smoking among boys aged 12 to 18 declined only from 14.7% to 10.7%.[12] In contrast, over the same period, smoking among all teenaged girls increased from 8.4% to 12.7%.[13] Indeed, among 17 to 18 year-olds, 26.2% of girls, as compared to 19.3% of boys, smoke, the only age level where female smokers outnumber males.[14] Overall, more teenagers are smoking today than ever and they are starting to smoke at a younger age.[15] This is especially significant because the earlier one begins to smoke, the stronger the habit is likely to become and the more difficult it will be to break.[16]

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[9] Office on Smoking and Health, U.S. Department of Health, Education and Welfare, The Health Consequences of Smoking for Women: A Report of the Surgeon General (1980) ("1980 Surgeon General's Report on Smoking and Women") at 23.

[10] Id., Preface at v.

[11] National Center for Health Statistics, Changes in Cigarette Smoking Practices Among Adults (1979) Table 2 at 2978.

[12] The National Institute of Education, U.S. Department of Health, Education and Welfare, Teenage Smoking: Immediate and Long-Term Patterns (1979) at 7.

[13] Id., at 8. Although smoking rates for teenaged girls overall have risen, smoking rates among girls aged 15 to 16 have decreased from 20.2% in 1974 to 11.8% in 1979.

[14] Id.

[15] 1980 Surgeon General's Report on Smoking and Women at 325.

[16] Id.

The decline in the percentage of adults who smoke has been accompanied by a rise in the number of cigarettes consumed per smoker, especially for female smokers.[17] In 1966, 34.7% of male smokers aged 21 and over smoked 25 or more cigarettes daily. In 1975, the percentage had increased to 36%.[18] Among female smokers 21 and over, the percentage who smoke 25 or more cigarettes daily rose from 16.9% in 1966 to 22.8% in 1975.[19]

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[17] 1979 Surgeon General's Report, Appendix at 17.

[18] Id., Appendix at 18, Table 8.

[19] Id.

II. HEALTH EFFECTS OF SMOKING

A. Introduction

The 1979 Surgeon General's Report concludes that "[c]igarette smoking is the single most important environmental factor contributing to premature mortality in the United States." [20] The 1979 Report, based on over 30,000 articles on smoking and health, confirms, strengthens and expands the conclusions of the 1964 Surgeon General's Report, which was the first comprehensive domestic report to conclude that smoking is a major threat to health.[21]

Since 1964, scientists and physicians have learned more about virtually every aspect of the adverse health effects of smoking, and the Surgeon General has issued seven additional reports on smoking and health. Researchers have learned much about several areas that received little attention in the 1964 Report but which are discussed in considerable detail in the 1979 Report, including the effects of maternal smoking during pregnancy on the fetus and newborn child, the development of lung cancer in women, the health effects of carbon monoxide and the interaction between smoking and birth control pills. Recent research has also strengthened the evidence demonstrating the relationship of smoking to lung and other cancers, heart attack and chronic obstructive lung disease. So much has been learned about smoking and health that the preface to the 1979 Surgeon

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[20] *Id*. Ch. 2 at 9 (emphasis added).

[21] *Id.*, Secretary's Forward at i.

General's Report declares "...the scientific evidence on the health effects of smoking is overwhelming."[22]

After more than 30 years of research, the evidence now shows that cigarette smokers suffer from significantly higher rates of death (mortality) and illness (morbidity) than nonsmokers.[23] Overall, a smoker is 70% more likely to die at a given age than is a comparable nonsmoker.[24] The increased death rate is even more dramatic when viewed in terms of life expectancy. On the average, a thirty year old heavy smoker (two or more packs a day) decreases his life expectancy by more than eight years.[25] Although people who smoke more are most likely to die at an early age, even males who smoke only one to nine cigarettes a day have substantially higher mortality rates than comparable nonsmokers.[26] Significantly, the data also reveal that the younger one starts smoking, the greater the risk of premature death[27] and the longer one smokes, the more that person increases the risk of dying prematurely as a result of smoking.[28]

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[22] Id., Preface at vii.

[23] Id., Ch. 2 at 12-15. During the past thirty years there have been eight massive prospective epidemiological studies that analyzed the effect of smoking on mortality or death rates. These eight studies, involving more than 16 million person years of experience, analyzed over 300,000 deaths.

Epidemiology is the science dealing with the factors that determine the frequency and distribution of disease in the population. Dorland's Medical Dictionary 459 (23rd ed. 1957).

Epidemiological studies may be prospective or retrospective. A prospective study is one which starts with a group of people who do not have the disease being studied. The group is divided into smokers and nonsmokers and then followed over a period of time to determine which people develop the disease. In a retrospective study, the researcher studies patients who already have the disease and a control group of people who do not have the disease and calculates the percentage of smokers in each group.

[24] 1979 Surgeon General's Report, Ch. 2 at 43.

[25] Id.

Many disease causing agents increase the likelihood of death from only one disease or affect only one bodily organ. By contrast, cigarette smoking increases the death rate for several diseases in various parts of the body. Heart disease is the chief contributor to premature death caused by smoking.[29] Lung cancer is second and chronic obstructive lung disease (emphysema and chronic bronchitis) is third.[30]

Smokers not only die younger than nonsmokers, they are also sick more often. Overall, smokers are more likely to have chronic conditions than nonsmokers.[31] Smokers miss more days of work due to injury and illness than do nonsmokers and spend more days in bed due to illness.[32] It is estimated that smoking causes a total loss of over 81 million work days each year, accounting for over 20% of all work days lost.[33] In the report of the National Commission on Smoking and Public Policy, the direct health care costs for 1975 due to smoking were estimated as between 11.1 and 11.4 billion dollars, while the 1977 figures were estimated at 15 billion dollars. A National Dilemma: Cigarette Smoking or the Health of Americans (1978) at 4.

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[26] Light smokers are 20%-45% more likely to die at a given age than are comparable nonsmokers. Id., Ch. 2 at 15-17.

[27] Id., Ch. 2 at 19-21.

[28] Id., Ch. 2 at 17-18.

[29] Id., Ch. 2 at 39.

[30] It is estimated that lung cancer will kill 101,000 people in 1980. 1980 Surgeon General's Report on Smoking and Women at 7. Eighty percent of these deaths are believed to be due to smoking. Holbrook, Tobacco Smoking, in Harrison's Principles of Internal Medicine, 960 (9th ed. K. Isselbacher, 1980).

There were 46,000 deaths from chronic obstructive lung disease in 1977. 1980 Surgeon General's Report on Smoking and Women at 160. Approximately 70% of these deaths are due to smoking. Holbrook, supra, at 940.

[31] 1979 Surgeon General's Report, Ch. 3 at 10.

The Surgeon General's Reports are not the only extensive evaluations of the medical evidence to conclude that cigarette smoking is a serious health hazard. The same conclusion has also been reached by virtually every major organization which has studied the issue, including the World Health Organization[34] and the National Commission on Smoking and Public Policy that was organized by the American Cancer Society.[35] For example, the National Commission concluded:

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[32] Id., Ch. 3 at 12.

[33] Id., Ch. 3 at 13.

[34] World Health Organization, Smoking and Its Effects on Health (Technical Series, No. 568, 1965).

[35] Report of the National Commission on Smoking and Public Policy, American Cancer Society, Inc., A National Dilemma: Cigarette Smoking or the Health of Americans (1978).

> Cigarette smoking remains the largest single unnecessary and preventable cause of illness and early death.[36]

Research sponsored by the tobacco industry also points to the dangers of smoking. In 1978, the American Medical Association Education and Research Foundation ("AMA-ERF") published a volume entitled Tobacco and Health that summarized almost ten years of research in the area of cigarette smoking and health that was financed by the cigarette manufacturers and supervised by the AMA-ERF. The 795 studies reported in this volume are almost exclusively clinical, rather than epidemiological, and are focused on laboratory examination of the effects of various constituents of cigarette smoke. The research did not focus on cancer because cancer research was being generously financed by other sources.[37] Nonetheless, based only on the research conducted under its own general direction, the AMA-ERF Committee for Research on Tobacco and Health concluded that the "tar" in cigarette smoke contains potent "co-carcinogens".[38] The AMA-ERF Committee, again basing its findings solely on the research produced under its direction, did address the association between smoking and chronic obstructive lung disease. It concluded:

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[36] Id., at 3. The National Commission's conclusion was an affirmation of the identical conclusion reached by the United States Public Health Service in its 1975 report entitled The Health Consequences of Smoking.

[37] American Medical Association Education and Research Foundation, Tobacco and Health (1978) at xiii.

[38] Id.

> Cigarette smoking plays an important role in the development of chronic obstructive pulmonary diseases and constitutes a grave danger to individuals with preexisting diseases of the coronary arteries.[39]

The medical evidence has also been confirmed by studies by the American insurance industry. Several life insurance companies explicitly recognize that nonsmokers are better health risks than smokers and charge higher premiums to smokers. One insurance company has published a detailed description of its experiences in offering nonsmokers preferred rates.[40] The company's report concludes:

> On the basis of our experience, we have confirmed that the mortality differentials between smokers and nonsmokers are large enough to validate the separate identification of these two groups for life insurance underwriting purposes.[41]

Thus, cigarette smoking indisputably is a major health hazard. The individual diseases associated with smoking are discussed in the sections that follow. As the discussion will illustrate, the overall effect of smoking is striking.

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[39] Id.

[40] Cowell, M.J. & Hirst, B.L., State Mutual Life Assurance Company of America, Mortality Differences Between Smokers and Nonsmokers (1979).

[41] Id., at 16.

B. Cancer

Cigarette smoking has been established as a major cause of cancer of the lung, larynx, oral cavity, esophagus, and bladder and is significantly associated with cancer of the pancreas and kidney.[42]

1. Lung Cancer

Lung cancer kills more people in the United States than does any other malignant disease.[43] Cigarette smoking is by far the most important cause of lung cancer in men and women, causing at least 80% of all lung cancer cases in the United States.[44] In 1980, lung cancer will cause an estimated 101,000 deaths, or over 275 deaths each day.[45] Of these deaths, more than 80,000 are attributable to lung cancer caused by cigarette smoking.[46] Moreover, persons who develop lung cancer have little chance of long-term survival. Seventy percent of lung cancer patients die within one year of diagnosis and 90% die within five years.[47]

The weight of the evidence establishing the relationship between smoking and lung cancer now "exceeds by several times" that available in 1964.[48] The 1964 Surgeon General's Report concluded that cigarette smoking was "causally related to lung cancer in men" and that the data for women "point[ed] in the same direction."[49] By 1979, it could be stated categorically that "[c]igarette smoking is the *major cause of lung cancer* in both men and women."[50] The 1980 Surgeon General's Report includes an excellent discussion of the relationship of smoking to the development of lung cancer in women. While it was once thought that women might be less susceptible to lung cancer than men, the data now reveal that differences in lung cancer rates between males and females have been due merely to differences in their smoking habits.[51] Recent years have seen a rapid increase in lung cancer rates among women, reflecting the fact that women first began to smoke in large numbers some 25 to 30 years after the increase in cigarette smoking among men. Currently, the rise in lung cancer death rates among women is "much steeper" than in men. At the current rates of increase, lung cancer death rates among women will surpass breast cancer (the number one cancer in women) death rates in the early 1980's.[52]

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[42] 1979 Surgeon General's Report, Ch. 5 at 31, 36, 42, 44, 49, 53.

[43] Id., Ch. 5 at 9.

[44] Id., Ch. 5 at 31. Holbrook, *supra*, at 940.

[45] 1980 Surgeon General's Report on Smoking and Women at 7 and Errata Sheet.

[46] Holbrook, *supra*, at 940.

[47] 1979 Surgeon General's Report, Ch. 5 at 31.

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[48] 1979 Surgeon General's Report, Ch. 5 at 31.

[49] U.S. Dept. of Health, Education, and Welfare, Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service (1964) ("1964 Surgeon General's Report").

[50] 1979 Surgeon General's Report, Ch. 5 at 31 (emphasis added).

[51] 1980 Surgeon General's Report on Smoking and Women at 131, 134. Smoking among women became prevalent more recently than among men, and women tended to smoke in ways that decreased the risk of lung cancer. That is, women tended to smoke fewer cigarettes per day, to inhale less smoke, and to smoke lower "tar" and nicotine, filter cigarettes. *Id.*, at 134-135.

The combined data from the eight largest prospective epidemiological studies help establish the carcinogenic effects of smoking[53] and reveal that "cigarette smokers on the average are 10 times as likely to develop lung cancer as nonsmokers."[54] For those who smoke two or more packs of cigarettes per day, the risk of lung cancer is twenty times that of the nonsmoker.[55]

The strong dose-response relationship between cigarette smoking and the development of lung cancer further establishes the causal relationship between smoking and lung cancer. The data reveal that the more one is directly exposed to cigarette smoke, the greater is that person's risk of developing lung cancer. For example, the number of cigarettes smoked per day, the number of puffs per cigarette, the depth of inhalation, and the age of initiation of smoking all have been shown to affect the risk of development of l cancer.[56]

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[52] *Id.*, at 152.

[53] These studies are described in a chart in the 1979 Surgeon General's Report, Ch. 2 at 13. The single most often-cited research effort establishing the carcinogenic effects of smoking is a prospective epidemiological study begun by the American Cancer Society (ACS) in 1959. This study enrolled more than 1,000,000 men and women, ranging in age from 30 to 100 and living in 25 states. Hammond, *Smoking in Relation to the Death Rates of One Million Men and Women* (National Cancer Institute Monograph No. 19). All segments of the population were included in the ACS study, except migrant workers and similar groups that could not have been traced over an extended period of time. The subjects completed a detailed questionnaire, including information concerning smoking habits. They were traced at regular intervals through 1972, and the oldest subjects still are being traced periodically, most recently in 1978. Hammond & Seidman, *Smoking and Cancer in the United States*, 9 Preventive Med. 171 (1980).

[54] 1979 Surgeon General's Report, Ch. 5 at 11.

[55] *Id.*, Ch. 5 at 12-13.

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[56] Id., Ch. 5 at 12.

The epidemiological evidence has been supported well by the d obtained from other kinds of research, including pathological stu and animal studies. In a well-known study conducted at the Veter Administration Hospital in East Orange, New Jersey, cross-section lung tissue from the lungs of men who had died from lung cancer a men who had died from other causes were analyzed in blind fashior Auerbach, Stout, Hammond, & Garfinkel, Changes in Bronchial Epithelium in Relation to Cigarette Smoking and in Relation to Lu Cancer, reprinted from 265 New England J. Med. 253-67 (1961). At same time, information about the smoking histories of these peopl was obtained from their families. When tissue analysis was combi with the data on smoking habits, it became clear that increased amounts of smoking were associated with "tremendous increase in t number of atypical [pre-cancerous] cells." Id. at 17. Thus, thi study strengthened the already overwhelming epidemiologic evidenc that cigarette smoking is a major cause of lung cancer. Id.

2. Other Cancers

The other cancers associated with cigarette smoking also kill a large number of people. In 1980, more than 23,000 people will die from smoking-related malignancies other than lung cancer, including cancer of the larynx, oral cavity, esophagus, urinary bladder, pancreas, and kidney.[57] Cigarette smokers have a laryngeal cancer mortality rate that is 6 to 13 times greater than that of nonsmokers.[58] For cancer of the oral cavity, mortality rates for smokers range from 3 to 10 times those of nonsmokers.[59] For smokers, the risk of esophageal cancer ranges from nearly twice to nearly 9 times that of the nonsmoker.[60] On the average, cigarette smokers are twice as likely to die from cancer of the urinary bladder

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[57] It has been calculated that cigarette smoking accounts for the following percentages of these cancer deaths: laryngeal 65%; esophageal 60%; oral 56%; bladder 30%; renal 30%; and pancreas 30%. (57 J. Nat'l Cancer Inst. 1207 (1976)). The American Cancer Society estimates for deaths from these cancers in 1980 (1980 Surgeon General's Report on Smoking and Women at 130) allow the following calculations:

	Total Deaths X % Attributable to Smoking	Total Deaths Attributable to Smoking
Larynx	3500 X 65%	2275
Esophagus	7600 X 60%	4560
Oral	8800 X 56%	4928
Bladder	10300 X 30%	3090
Renal	7900 X 30%	2370
Pancreas	20900 X 30%	6270

[58] 1979 Surgeon General's Report, Ch. 5 at 33.

[59] *Id.*, Ch. 5 at 39-40.

[60] *Id.*, Ch. 5 at 42-43.

as nonsmokers.[61] Moreover, known specific carcinogens for the human bladder have been identified in cigarette smoke.[62] With respect to cancer of the kidney, mortality rates for cigarette smokers vary from 1-1/2 to 2-1/2 times those of nonsmokers.[63] Finally, with regard to pancreatic cancer, the mortality rate is approximately twice as high for cigarette smokers as for nonsmokers.[64] Significantly, a dose-response relationship with cigarette smoking has now been established for all of these forms of cancer.[65] Thus, heavy smokers incur risks substantially greater than the average risks noted above.

As with lung cancer, the conclusions regarding smoking and cancer of the larynx, esophagus, and oral cavity have been significantly strengthened and expanded since 1964.[66] Moreover, the association between cigarette smoking and cancer of the pancreas[67] and kidney[68] is based on new evidence, not reported in the 1964 Surgeon General's Report. In addition, the 1979 report establishes that there is a synergistic relationship between cigarette smoking and use of alcohol that makes those who both smoke and drink alcoholic beverages heavily far more likely to develop cancer of the larynx, oral cavity, and esophagus than those who consume either cigarettes or alcoholic beverages alone.[69]

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[61] Id., Ch. 5 at 45.

[62] Id., Ch. 5 at 47.

[63] Id., Ch. 5 at 48.

[64] Id., Ch. 5 at 50.

[65] Id., Ch. 5 at 37, 42, 44, 49, 53.

[66] Id., Ch. 5 at 36, 42, 44.

[67] Id., Ch. 5 at 53.

[68] Id., Ch. 5 at 49.

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[69] Id., Ch. 5 at 37, 42, 45.

C. *Heart Disease* [70]

Coronary heart disease (CHD) accounts for nearly one half of the deaths in this country.[71] Heart attack - the most dramatic manifestation of coronary heart disease - is the single greatest cause of death in the United States.[72] There is no longer any doubt that cigarette smoking is an important "risk factor" for heart disease - that is, people who smoke significantly increase their chances of suffering from heart disease in the future.[73] Smoking is one of three *major* risk factors for heart attack, and no risk factor is more important.[74] In fact, cigarette smoking annually accounts for nearly one-third of all the deaths from coronary heart disease. The Surgeon General estimated that in 1978 alone, 225,000 CHD deaths were attributable to cigarette smoking.[75]

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[70] Cigarette smoking has also been linked to other types of cardiovascular disease, including peripheral vascular disease and stroke. For a discussion of the relationship between smoking and peripheral vascular disease, *see* 1979 Surgeon General's Report, Ch. 4 at 52-55. For a discussion of the relationship between smoking, birth control pills and stroke, *see* Section G, *infra*.

[71] American Heart Association, Heart Facts, (1978) at 2.

[72] *Id.*, at 5. *See, also, National Commission on Egg Nutrition, et. al.*, 88 F.T.C. 89, 119 (1976) *modified*, 570 F.2d 157 (7th Cir. 1977) *cert. denied* 439 U.S. 821 (1978).

[73] 1979 Surgeon General's Report, Ch. 4 at 35; American Heart Association, Heart Facts, *supra* at 15. The precise mechanism by which cigarette smoking increases the risk of heart attack or other manifestations of heart disease has yet to be identified. Many experts believe that either the carbon monoxide or the nicotine in cigarettes is primarily responsible for the increased risk, but neither has been shown conclusively to be the responsible agent. 1979 Surgeon General's Report, Ch. 4 at 36-39.

[74] 1979 Surgeon General's Report, Ch. 2 at 41. The two other major risk factors are hypertension and high blood cholesterol.

The evidence documenting the role of cigarette smoking as a major risk factor for CHD is now overwhelming.[76] As the Surgeon General noted, "[s]ystematic observations on the association between smoking and cardiovascular diseases have been made in considerably more than a million individuals in the United States alone and have involved many millions of person years of experience."[77]

Based on this voluminous evidence, the following facts can be stated conclusively concerning the relationship between smoking and cardiovascular disease:

1. Smoking nearly doubles one's risk of heart attack and other coronary heart disease.

Many age-adjusted studies consistently show that smoking increases the risk of heart attack or other coronary heart disease by a factor of two: i.e., smoking doubles the risk of suffering from CHD. The increased risk of coronary heart disease caused by smoking is greater for young people than older people,[78] greater for Americans than for people of some other countries,[79] and is independent of other risk factors of CHD, such as high blood cholesterol levels and high blood pressure.[80] The relationship between coronary heart disease and smoking exists when either deaths or total CHD events are analyzed, and remains remarkably constant across the various studies performed in this country.[81] The data on women is not as extensive as the data on men, but several studies of coronary heart disease in women indicate that the heart disease risk for women smokers is also about twice that of their non-smoking counterparts.[82]

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[75] 1979 Surgeon General's Report, Secretary's Forward at ii.

[76] The Commission has previously recognized that smoking is a major risk factor for CHD. See, National Commission on Egg Nutrition, 88 F.T.C. at 120, finding of fact by ALJ #54, adopted in Commission Opinion at 201-02.

[77] 1979 Surgeon General's Report, Ch. 4 at 63.

[78] Id., Ch. 4 at 35.

[79] Some studies in other countries indicate a less important role for cigarette smoking in contributing to CHD. For example, a study (Continued)

A few of the major studies which document the increased risk of CHD attributable to smoking are summarized below:[83]

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[79] (Footnote Continued)

of Japanese smokers revealed only a slight increase of CHD risk over that of nonsmokers. Hirayama, 1967, summarized in 1979 Surgeon General's Report, Ch. 4 at 24. This is most likely explained by the fact that the Japanese have such low blood cholesterol levels that heart attacks from all causes are relatively rare among Japanese, and in any event does not at all modify the conclusion that smoking is a major cause of CHD *in the United States*. 1979 Surgeon General's Report, Ch. 4 at 37.

[80] Studies of Americans which hold constant other major risk factors, such as blood cholesterol level and blood pressure, find cigarette smoking a powerful predictor of CHD. *See, e. g.*, the Framingham Heart Study, summarized by Doyle, *et. al.*, 1964, cited in 1979 Surgeon General's Report, Ch. 4 at 22, 37.

[81] 1979 Surgeon General's Report, Ch. 4 at 65.

[82] 1980 Surgeon General's Report on Smoking and Women at 102.

[83] Data obtained from 1979 Surgeon General's Report, Ch. 4 at 22-25.

STUDY	SAMPLE	FINDINGS
Hammond and Horn, 1958	190,000 males, 50-64 years of age in 9 states	Smokers had 1.7 times the risk of death from heart disease of nonsmokers. Smokers of one to two packs a day had 2.2 times the risk; two pack a day or more smokers had 2.4 times the risk.
Doyle, et. al., 1964	4,000 males, Framingham, MA, Albany, NY 30-62 years of age.	All smokers, 2.4 times the risk of death from heart disease than nonsmokers. More than a pack a day smokers had 3.5 times the risk of death.
Pooling Project, American Heart Association, 1970	7,400 males, 30-59 years of age.	One pack a day smokers 1.7 times the risk; more than one pack a day smokers had 3 times the risk of death from heart disease than nonsmokers.
Kannel, et. al., 1968	5,000 males and females, 30-59 years of age.	Male smokers had 1.5 times the risk of suffering a heart attack than nonsmokers, females had a relative risk of 1.7.
Shapiro, et. al., 1969	110,000 males and females, 35-64 years of age.	Both males and females had twice the risk of suffering from CHD if they smoked. For two packs or more a day smokers, the risk was about 6 times that of a nonsmoker.

2. The more cigarettes smoked, the greater the risk of CHD.

One of the most striking characteristics about the relationship between smoking and heart disease is that it is dose-responsive; heavier smokers run a much greater risk than lighter smokers, and lighter smokers run a greater risk than nonsmokers. The dose-responsive nature of the smoking--heart disease relationship is evident from the chart above: Hammond and Horn reported a relative risk (compared to nonsmokers) of 1.7 for all smokers, 2.2 for one pack a day smokers, and 2.4 for two pack a day smokers. Both Doyle, *et. al.*, and the Pooling Project found that people who smoked more than a pack a day had *three* times the risk of nonsmokers, and a considerably greater risk than lighter smokers. Two pack a day smokers were about *six times* more likely to suffer a heart attack than nonsmokers in the Shapiro study. The dose-response nature of the relationship between cigarette smoking is also an important element in establishing that smoking *causes* heart disease.[84]

3. The risk of CHD decreases if smoking is stopped.

Another important element in determining the link between coronary heart disease and smoking is what epidemiologists refer to as "cessation" data: what happens to people when they stop smoking? The evidence strongly indicates that cessation of smoking will reduce the risk of heart disease. Two major studies illustrate this point. Hammond and Garfinkle, 1969, reported that while current smokers had nearly double the risk of heart attack of nonsmokers, ex-smokers suffered only a slightly greater risk than nonsmokers.[85] Among heavy smokers, those currently smoking had twice the risk of heart attack than those who had quit.[86] The Pooling Project study found that ex-smokers had virtually the same risk of CHD death as nonsmokers, while smokers' risk was 1.7 times greater, and 3 times greater for smokers of more than a pack a day.[87]

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[84] 1979 Surgeon General's Report, Ch. 4 at 66.

4. Conclusion

The Surgeon General summarized the massive data linking smoking and heart disease as follows:

> In industrialized societies which share about the same general nutritional and metabolic circumstances as the United States, it has been shown repeatedly that cigarette smoking is associated with myocardial infarction [heart attack] and death following infarction when compared to the risk among nonsmokers. The effect is dose-related in terms of years of smoking, number of cigarettes smoked per day, and the habit of inhaling. The association is generally consistent, reproducible and predictive. It is independent in the sense that its effect is found when other risk factors for heart disease are controlled in statistical analysis...Cessation of smoking reduces, over time, the increased risk attributable to smoking toward the risk of nonsmokers. 1977 Surgeon General Report, Ch. 4 at 35.

The Surgeon General's Report carefully considered the issue of causation with respect to smoking and heart disease in 1979. It acknowledged that correlation is not synonymous with causation. It considered the major argument raised against the conclusion that causation has been demonstrated, and, based on the massive evidence, rejected that argument.[88] Ultimately, the Report concluded that:

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[85] Smokers had a 90% increased risk over nonsmokers; ex-smokers had about a 15% increased risk. 1979 Surgeon General's Report, Ch. 4 at 34.

[86] 1979 Surgeon General's Report, Ch. 4 at 34.

[87] *Id.*

> Given the characteristics of its association with heart attack (such as strength, graded relationship, independence, consistency, antecedence, loss of relationship on withdrawal, predictive capability, and a degree of coherence), it can be concluded that smoking is causally related to coronary heart disease in the common sense of that idea and for purposes of preventive medicine. 1979 Surgeon General's Report, Ch. 4 at 66.

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[88] It has been suggested that the increased incidence of heart disease among smokers might be explained by individual characteristics of smokers, rather than smoking itself. In other words, the argument runs, people who smoke also coincidentally are people more likely to have a heart attack, but smoking isn't actually related to heart attack. This is the so-called "constitutional" argument: smokers are more prone to heart attack than nonsmokers, because of underlying constitutional differences between the groups. See, e.g., Seltzer, Smoking and Coronary Heart Disease: What Are We To Believe? 100 American Heart Journal 275 (1980). However, proponents of this argument have never been able to identify any characteristic of smokers that might account for this increased risk, and studies which control for known risk factors demonstrate smoking acts independently of other factors to cause heart disease. Moreover, the constitutional hypothesis is logically inconsistent with the established dose-response relationship between smoking and heart disease, and the reduction in risk of heart disease among people who quit smoking. For example, the reduction in risk upon cessation "is contrary to the constitutional concept as expressed above, unless further complex assumptions are made and it is assumed that large numbers of individuals underwent a change in their underlying constitutional factor in midlife, acquired low risk, and ceased to smoke because of that new constitution." Thus, the Surgeon General concluded, based on all the evidence, that "the constitutional hypothesis... does not provide a credible basis to doubt that cigarette smoking is a cause of coronary heart disease." 1979 Surgeon General's Report, Ch. 4 at 66.

D. Chronic Obstructive Lung Disease (Emphysema and Chronic Bronchitis)

1. Mortality and morbidity

Cigarette smoking is the major cause of emphysema and chronic bronchitis.[89] These disorders reduce the lungs' ability to inhale oxygen and to exhale carbon dioxide.[90] At their early stages the most important symptoms of these diseases may be a slight reduction in exercise capacity.[91] As they progress, these disorders make breathing increasingly difficult.[92] In their advanced stages, they can cause death.[93]

Chronic bronchitis and emphysema are frequently difficult to distinguish and usually coexist in the same person.[94] Consequently, the two disorders are collectively labeled "chronic obstructive lung disease" ("COLD") or "chronic airflow obstruction."[95] These terms accurately describe the symptoms of both diseases and make it unnecessary to identify them separately.

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[89] 1979 Surgeon General's Report, Ch. 2 at 41.

[90] Unlike lung cancer, these disorders are not malignant. The non-malignant lung diseases are also called non-neoplastic bronchopulmonary diseases.

[91] Ingram, *Chronic Bronchitis, Emphysema, and Chronic Airway Obstruction*, in *Harrison's Principles of Internal Medicine*, 1238-39 (9th ed. K. Isselbacher, 1980).

[92] *Id.*

[93] *Id.*

[94] 1979 Surgeon General's Report, Ch. 6 at 8.

[95] *Id.* Although the symptoms of chronic bronchitis and emphysema are the same--an obstruction in expiratory flow accompanied by oxygen deficiency during exertion--the cause of the symptoms is different. Emphysema is characterized by a breakdown in the walls of the (Continued)

Chronic obstructive lung disease kills thousands of smokers each year. It is conservatively estimated that there were more than 34,000 smoking related deaths from chronic obstructive lung disease in 1979.[96] Numerous prospective and retrospective epidemiological studies have established that smokers are more likely than nonsmokers to develop chronic obstructive lung disease.[97] Six prospective studies representing a total of over 13 million patient years of observation and almost 270,000 deaths from all causes found that smokers were from 2.3 to 24.7 times more likely to die of chronic obstructive lung disease than were nonsmokers.[98] While the mortality ratios in these studies vary considerably, they all lead to

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[95] (Footnote Continued)

alveoli, the tiny airsacks in the lungs from which oxygen is transferred to the blood and carbon dioxide is removed from the blood. The breakdown in the alveolar walls reduces the surface area of the lungs that is exposed to the bloodstream and diminishes the lungs' ability to deliver oxygen and remove waste gases. A conclusive diagnosis of emphysema can only be made after death through autopsy.

Chronic bronchitis can be diagnosed prior to death and is characterized by two main dysfunctions: (1) fixed narrowing of the airways and (2) mucous formation in the airways. If someone suffers from narrowing of the airways and extra production of mucous, both of which may be found by specialized tests of pulmonary function, for more than three months a year, two years in a row, that person is characterized as having chronic bronchitis.

[96] See 1980 Surgeon General's Report on Smoking and Women at 160 in which it is reported that a total of 46,000 people died of COLD in 1977; n. 100, _infra_, where it is estimated that 70-80% of those deaths are smoking related.

[97] 1979 Surgeon General's Report, Ch. 6 at 9.

[98] _Id._, Ch. 6 at 10.

the same conclusion: smokers are far more likely to die from COLD than are nonsmokers.[99] In fact, experts estimate that 70%-80% of the chronic obstructive lung disease deaths each year are smoking related.[100]

There is also a strong dose-response relationship between smoking and death rates from chronic obstructive lung disease. The more one smokes, the more likely one is to die from emphysema or chronic bronchitis. One major study may be used to illustrate this dose-response relationship. A 20 year follow-up study of over 34,000 British physicians projected that light smokers (1-14 cigarettes per day) have an annual death rate from chronic obstructive lung disease of over 10 times the rate for nonsmokers while heavy smokers (25 or more cigarettes per day) have an annual chronic obstructive lung disease death rate of over twice that of light smokers.[101] Medium smokers (15-24 cigarettes per day) were projected to have a COLD death rate between the death rate of light and heavy smokers.[102]

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[99] The variation in mortality rates may result from differences in diagnostic methods and an underestimation of deaths due to COLD. Id., Ch. 6 at 9.

[100] Holbrook, Tobacco Smoking, in Harrison's Principles of Internal Medicine, 940 (9th ed. K. Isselbacher, 1980).

[101] The study projected annual death rates of 88 per 100,000 for heavy smokers; 50 per 100,000 for medium smokers; 38 per 100,000 for light smokers; and 3 per 100,000 for nonsmokers. 1979 Surgeon General's Report, Ch. 6 at 10.

[102] Id.

Increased mortality is not the only problem caused by chronic obstructive lung disease. Chronic obstructive lung disease develops and progresses slowly, but continuously, over a period of years, and exacerbates the normal decline in pulmonary function that occurs with age. Because the symptoms of chronic obstructive lung disease develop over a long period of time and increase with age, chronic obstructive lung disease is usually a gradually incapacitating disorder, rather than a quickly fatal one. Its victims often suffer long term illness, disability or hospitalization before they die from chronic obstructive lung disease or other causes.[103] In fact, chronic obstructive lung disease is second only to coronary heart disease as a cause of Social Security disability compensation.[104]

Some of the effects of chronic obstructive lung disease are irreversible -- the breakdown of the alveolar walls in emphysema cannot be reversed.[105] One who stops smoking after developing the symptoms of chronic obstructive lung disease will almost immediately stop the acceleration of the development of chronic obstructive lung disease symptoms and may have some improved pulmonary function, but will not improve to his/her age-adjusted normal range.[106] In effect, chronic obstructive lung disease speeds one's normal age related decline in respiratory function. As a result, persons who have developed severe symptoms of chronic obstructive lung disease from smoking greatly increase their risk of dying of chronic obstructive lung disease even after they quit smoking. The authors of the British physicians study discussed above suggest that many of the ex-smokers they studied who died of chronic obstructive lung disease within 5-9 years of quitting, died because the irreversible decrease in lung function caused by smoking was worsened by the natural decline caused by age.[107]

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[103] Id.

In addition, it is well-established that smokers suffer from more minor respiratory symptoms than nonsmokers, including cough, sputum production and wheezing. These relationships are also dose-responsive. Id., Ch. 6 at 20.

[104] Id., Ch. 6 at 7.

[105] Id., Ch. 6 at 10.

[106] Id., Ch. 6 at 22-23.

2. The role of smoking in the development of chronic obstructive lung disease

Retrospective and prospective epidemiological studies have solidly established that smoking causes chronic obstructive lung disease.[108] Pathological studies have confirmed that there is a strong dose-response relationship between smoking and alveolar damage.[109] In addition, both clinical and pathological studies have linked smoking to functional lung changes such as small airways dysfunction, which may be a precursor of emphysema.[110]

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[107] 1979 Surgeon General's Report, Ch. 6 at 10.

[108] Id., Ch. 2 at 4; Ch. 6 at 9.

[109] Id., Ch. 6 at 23-24.

Despite these findings, physicians and scientists do not know exactly how chronic obstructive lung disease develops. Researchers have proposed and found support for three mechanisms by which smoking might lead to the development of chronic obstructive lung disease. These include (1) altering the protease-anti-protease balance in the lungs, (2) compromising immune mechanisms, and (3) interfering with pulmonary clearance mechanisms. 1979 Surgeon General's Report, Ch. 6 at 25-33.

Some experts suspect that the irritating gases in cigarette smoke (Continued)

Smoking may act synergistically with certain occupational exposures to increase the risk of chronic obstructive lung disease. A number of studies have found that workers exposed to cotton fibers, asbestos, granite dust, and coal dust increase their incidence of chronic obstructive lung disease.[111] When persons who are regularly exposed to these substances also smoke, they further increase their risk of developing chronic obstructive lung disease.[112]

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109 (Footnote Continued)

contribute to the destruction of the alveolar walls as well as the cough and other signs of irritation found in chronic bronchitis. They believe that particulate matter in the "tar" in smoke contributes to the production of mucous and thickening of the lung membranes.

[110] Id., Ch. 6 at 12-19, 24.

[111] 1979 Surgeon General's Report, Ch. 6 at 7, 36.

[112] Id., Ch. 7 at 36.

E. Carbon Monoxide

Carbon monoxide ("CO") is now known to be one of the most harmful ingredients of cigarette smoke. Carbon monoxide, of course, is not a specific disease caused by smoking, but is a constituent of cigarette smoke that has been linked to many of the health hazards associated with smoking. Carbon monoxide is discussed here because, along with "tar" and nicotine, CO is now known to be an extremely harmful constituent of cigarette smoke, particularly with respect to maternal smoking during pregnancy, which is discussed in the next section.

Although CO was not even discussed in the 1964 Surgeon General's Report, the 1979 Surgeon General's Report identifies CO in cigarette smoke as a

> [P]ossible critical factor in coronary heart disease, atherosclerosis and sudden death, occupationally-related illness, chronic respiratory disease, fetal growth retardation, and the noxious effects of passive smoking.[113]

Carbon monoxide causes adverse health effects by reducing the blood's ability to carry oxygen to the cells. Hemoglobin, the compound in red blood cells that carries oxygen, combines more readily with CO than it does with oxygen. Thus, when CO combines with the hemoglobin in some red blood cells to form carboxyhemoglobin (COHb), there are fewer red blood cells available to deliver oxygen to the system.

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[113] 1979 Surgeon General's Report, Preface at xiii. In addition to "tar," nicotine and CO, cigarette smoke has about 2,000 constituents, and although less is known about them, many of these other compounds are also thought to be hazardous. Id., Ch. 1 at 29.

A nonsmoker is likely to have a carboxyhemoglobin level of about 1%.[114] In contrast, the average smoker has a COHb level of about 5%, and some one pack-a-day smokers may have COHb levels as high as 15%.[115] Individual smoking habits, such as depth and frequency of inhalation, also affect how much a cigarette will raise the carboxyhemoglobin level, but on the average each cigarette raises the COHb level by about .75%. The half-life of CO in the blood is about three to four hours, so after four hours the excess COHb caused by one cigarette will be reduced to somewhat less than .37%.[116] The length of time between cigarettes and the frequency, depth and total number of inhalations affect what the smoker's maximum daily COHb level will be.

The effects of CO on smokers fall into two general categories. First, there are acute effects -- the effects that occur immediately or soon after an individual smokes a cigarette. The second type involves the effect of CO in the pathogenesis or development of diseases associated with smoking. While acute effects may be noticed almost immediately, the pathogenesis of disease occurs over a long period of time.

1. Acute Effects

Carbon monoxide in the levels found in cigarette smoke presents the greatest danger to a person for whom low level oxygen deprivation is hazardous. Thus, carbon monoxide exacerbates the symptoms of heart disease, chronic obstructive lung disease and anemia by reducing the delivery of oxygen to the system.[117] The particular cardiovascular diseases aggravated by CO include coronary heart disease, atherosclerosis, angina pectoris, cerebrovascular disease and peripheral vascular disease.[118] CO in cigarettes can also add to the already increased carbon monoxide levels of industrial workers or other people who are exposed to CO in the workplace or at other locations.[119]

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[114] Holbrook, Tobacco Smoking, in Harrison's Principles of Internal Medicine, 938 (9th ed. K. Isselbacher, 1979).

[115] Id., at 938-39.

[116] Drill, Pharmacology in Medicine, 936 (3rd ed. 1965).

CO in cigarette smoke is especially harmful to the developing fetus. Its effects are both acute in the sense that they are expressed relatively quickly, and long term, in that they may alter the outcome of pregnancy or the health of the child. Many, if not most, of the instances of perinatal death, complications of pregnancy and long term developmental problems that are associated with smoking are thought to result, at least in part, from the effects of CO on the fetus.[120] Carbon monoxide in cigarette smoke reduces the oxygen-carrying capacity of the blood of both the mother and fetus.[121] Compounding this effect is carbon monoxide's action to reduce the pressure at which oxygen is delivered to the fetal cells.[122] Thus, not only does CO make the blood less able to carry oxygen, it also reduces the percentage of available oxygen that is actually delivered to the fetus. As a result, the developing fetus receives less of the oxygen that it needs for its cells to multiply and grow and for the pregnancy to conclude successfully.[123]

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[117] 1979 Surgeon General's Report, Ch. 1 at 14, Ch. 11 at 34.

Aronow, Effects Of Passive Smoking On Angina Pectoris. 229 New England J. Med. 21-24 (1978).

Aronow, Ferlinz, & Glauser, Effects of Carbon Monoxide on Exercise Performance in Chronic Obstructive Pulmonary Disease, 63 Am. J. Med. 904-08 (1977).

[118] 1979 Surgeon General's Report, Ch. 4 at 47, 50; Ch. 11 at 27-28, 30, 34.

[119] 1979 Surgeon General's Report, Ch. 1 at 20; Ch. 7 at 8.

The effects of CO on people who are in good physical condition and are not pregnant have not been as well-established. There is evidence that levels of CO in the amount found in cigarette smoke decrease exercise performance.[124] In addition, some studies indicate that even low levels of CO may produce a slight deterioration in psychomotor performance, especially attentiveness and cognitive function.[125]

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[120] See, Longo, The Biological Effects of Carbon Monoxide on the Pregnant Woman, Fetus, and Newborn Infant, 129 Am. J. of Obstetrics and Gynecology 69-103 (1977).

[121] 1979 Surgeon General's Report, Ch. 1 at 20, Ch. 7 at 8.

[122] Id., Ch. 1 at 21-22, Ch. 8 at 70-72. 1980 Surgeon General's Report on Smoking and Women at 9, 229, 272-75.

[123] Numerous studies have shown that the ratio of placental weight to birth weight in babies of mothers who smoke is higher than for babies of nonsmokers. 1979 Surgeon General's Report, Ch. 8 at 11-17. Studies in animals have confirmed that carbon monoxide in the levels found in cigarette smoke reduces the oxygen carrying capacity of the blood and the partial pressure of oxygen in fetal blood, as well as increases the placental ratio. Id., Ch. 8 at 60-65. It is theorized that the increased weight of the placenta and reduced weight of the fetus in smoking mothers result from the system's response to relative fetal hypoxia (lack of oxygen). Id., Ch. 8 at 17. If the mother's system is unable to fully compensate for the reduced fetal supply of oxygen, it would not be surprising to see long term effects in the child's growth and development, as well as increased perinatal and neonatal death.

2. Pathogenesis

Carbon monoxide not only aggravates the symptoms of cardiovascular disease, it has also been cited as a factor in the development of coronary heart disease in the first place.[126] However, because coronary heart disease is caused by many factors and develops slowly, it is easier to identify cigarettes as a risk factor for coronary heart disease than to isolate CO as the causative agent.

3. Conclusion

Carbon monoxide in cigarette smoke is a serious health hazard. CO is harmful to anyone who has heart or lung disease or for whom a lowered oxygen level in the blood is dangerous. The developing fetus is particularly susceptible to the harms of low-level oxygen deprivation caused by carbon monoxide in cigarette smoke. It is possible that CO not only is dangerous to people with heart disease, but also is a factor in first causing heart disease.

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[124] 1979 Surgeon General's Report, Ch. 11 at 27.

[125] Id., Ch. 11 at 34.

[126] Aronow, Effects of Cigarette Smoking and of Carbon Monoxide on Coronary Heart Disease, in Smoking and Arterial Disease (Greenhalgh ed. 1980).

F. Smoking and Pregnancy

When the 1964 Surgeon General's Report on Smoking and Health was published, relatively little was known about the effects of maternal smoking on the fetus. In fact, the 1964 Report contained less than one page of discussion on the possible effects of smoking during pregnancy on the fetus and newborn infant. During the following years, physicians and scientists greatly increased their research in this area. The 1979 Surgeon General's Report and the 1980 Surgeon General's Report on Smoking and Women both contain lengthy scientific discussions of the effects on the fetus and newborn infant caused by smoking during pregnancy. The new conclusions reached in these discussions are summarized succinctly and unequivocally in the 1980 Report: "...cigarette smoking is a major threat to the outcome of pregnancy and the well being of the newborn baby."[127]

1. Fetal and infant mortality

Maternal smoking during pregnancy has been associated with several causes of fetal and perinatal death, including spontaneous abortion,[128] stillbirth,[129] respiratory distress syndrome,[130] pneumonia,[131] sudden infant death syndrome,[132] death from prematurity and unknown causes,[133] and with an increased incidence of preterm deliveries.[134] The relationship of smoking to these causes of fetal and infant death is affected by other factors that increase the risk of complications of pregnancy and may vary according to the mother's age, socio-economic status, race, or other factors.[135] Although these variations make the relationship subject to some controversy, the relationship is sufficiently strong that the 1979 Surgeon General's Report concluded: "...maternal smoking can be a direct cause of fetal or neonatal death in an otherwise normal infant."[136]

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[127] 1980 Surgeon General's Report on Smoking and Women, Preface at 1.

[128] 1979 Surgeon General's Report, Ch. 8 at 30-32.

[129] Id., Ch. 8 at 32.

[130] Id.

[131] Id.

[132] Id., Ch. 8 at 44.

The fetal and infant health problems associated with maternal smoking during pregnancy are labeled according to the length of time the complication or death occurs following conception. During the first 20-22 weeks of gestation, death of the fetus occurs as a result of a spontaneous or naturally occurring abortion. The period beginning 20-22 weeks after conception and continuing until four weeks after birth is called the perinatal period. Perinatal death occurring prior to successful delivery is known as a stillbirth. Perinatal death that occurs within four weeks of an infant's birth is called neonatal death.

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[133] Id., Ch. 8 at 47.

[134] Id., Ch. 8 at 42.

[135] Id., Ch. 8 at 28-29.

[136] Id., Ch. 8 at 47.

a. Spontaneous abortion

Several prospective and retrospective studies have found that smoking during pregnancy increases the risk of spontaneous abortion and that the risk increases with the amount smoked.[137] Spontaneous abortions are difficult to study because of problems in ascertaining whether a spontaneous abortion has actually taken place.[138] But the studies that have been completed have been sufficient to lead the Surgeon General's Reports to conclude that smoking during pregnancy significantly increases the risk of spontaneous abortion.[139]

b. Perinatal mortality

(Stillborn and Neonatal Death)

Smoking during pregnancy also increases the risk of perinatal death.[140] Although the increased risk is greatest for the developing fetuses or newborn children of women who also have an increased risk of perinatal death for other reasons, including low socio-economic class, low education level, less than optimum maternal age or race,[141] all women who smoke during pregnancy increase the risk of perinatal mortality.[142] After adjusting the rates of perinatal mortality for the effects of all other factors, an analysis of one large study found that light smokers (less than one pack per day) increase the risk of perinatal death by 20% and heavy smokers (more than one pack per day) increase the risk by 35%.[143] This relationship, confirmed by other studies, is highly significant, independent of other factors and dose-response related.[144]

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[137] 1979 Surgeon General's Report, Ch. 8 at 30.

[138] *Id.*

[139] *Id.*, Ch. 8 at 32; 1980 Surgeon General's Report on Smoking and Women at 243-45.

[140] 1979 Surgeon General's Report, Ch. 8 at 46.

[141] *Id.*, Ch. 8 at 33.

[142] *Id.*, Ch. 8 at 32-35.

Neonatal deaths of infants born to mothers who smoke result in part from complications associated with prematurity. Smoking significantly increases the risk of preterm delivery. Mothers who smoke have been found to be 36%-47% more likely to give birth prematurely.[145] Additionally, the few studies of the association between smoking and "sudden infant death syndrome" (SIDS) have all found that maternal smoking during pregnancy is positively associated with SIDS.[146]

2. *Low birth weight*

Smoking during pregnancy retards fetal growth and decreases infant birth weight.[147] Babies born to women who smoke during pregnancy weigh an average of 200 grams (about half a pound) less than babies born to comparable women who did not smoke during pregnancy.[148] This relationship has been found in almost 50 retrospective and prospective studies involving more than half a million total births.[149]

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[143] Meyer, Jonas, & Tonascia, *Perinatal Events Associated With Maternal Smoking During Pregnancy*, 103 Am. J. of Epidemiology 464-76 (1976).

[144] 1979 Surgeon General's Report, Ch. 8 at 47. The complications of pregnancy associated with smoking that contribute to the increased risk of perinatal death include placenta previa, abruptio placenta, bleeding during pregnancy and premature rupture of membranes. *Id.*, Ch. 8 at 39. Smoking appears to reduce the incidence of preeclampsia during pregnancy but also to greatly increase the risk of perinatal mortality if preeclampsia does develop. *Id.*, Ch. 8 at 41-42.

[145] *Id.*, Ch. 8 at 42.

[146] *Id.*, Ch. 8 at 44.

[147] *Id.*, Ch. 8 at 11-12.

Perhaps the most significant aspect of the relationship between maternal smoking and decreased birth weight is its strong linkage to the amount of smoking during pregnancy coupled with its independence from other factors that influence birth weight. That is, there is a dose-response relationship between maternal smoking and birth weight - the more a woman smokes during pregnancy, the greater the reductio in birth weight[150] -- and this reduction is independent of socioeconomic status, race, parity (number of children), age, maternal size, maternal weight gain, or sex of the child.[151]

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[148] Id., Ch. 8 at 11; 1980 Surgeon General's Report on Smoking and Women at 224.

[149] 1979 Surgeon General's Report, Ch. 8 at 11.

[150] Id., Ch. 8 at 12.

[151] Id., Ch. 8 at 12-13.

The independence of smoking from other factors that influence birth weight means that smoking during pregnancy affects all births similarly. Babies born to a woman who smokes during pregnancy will weigh an average of 200 grams less than would otherwise be expected even if the mother has no other characteristics that would be expected to reduce birth weight. Id., Ch. 8 at 14. Women who have other characteristics that would be expected to reduce their child's birth weight will reduce the child's birth weight by an additional (Continued

Not surprisingly, babies born to smoking mothers are not only lighter, but smaller in other dimensions as well. The fetal growth retardation associated with smoking may also be seen in reduced body length, chest circumference, and head circumference.[152] One of the major studies of maternal smoking, the Ontario Perinatal Mortality study of more than 50,000 births, found that, compared to nonsmokers, women who smoke a pack or more of cigarettes a day are about 130% more likely to have a baby that weighs less than the mean of 2500 grams and smokers of less than a pack a day are about 53% more likely to have babies that weigh less than the mean.[153]

There have been several studies of the effects of smoking during pregnancy and reduced birth weight on the long term physical growth, intellectual development and behavioral characteristics of the child. Such studies are difficult to conduct because of the large

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[151] (Footnote Continued)

200 grams if they smoke during pregnancy. Id.

Although there is a clear dose-response relationship between smoking during pregnancy and reduced infant birth weight, there is some evidence that women who stop smoking by the fourth month of pregnancy have about the same risk of giving birth to a low-birth weight baby as nonsmokers. Id., Ch. 8 at 12.

[152] 1980 Surgeon General's Report on Smoking and Women at 231.

The overall reduction in size of smokers' babies cannot be explained by the greater likelihood of smokers to deliver their babies prematurely and therefore to have smaller babies. Although, as discussed above, smokers do have more preterm deliveries, smokers' babies are smaller than nonsmokers' babies when matched for time of gestation. 1979 Surgeon General's Report, Ch. 8 at 28.

[153] 1979 Surgeon General's Report, Ch. 8 at 14.

number of factors other than maternal smoking during pregnancy that might affect the variables being studied. According to the 1979 and 1980 Surgeon General's Reports, several studies show that children whose mothers smoked during pregnancy display significant signs of retardation and growth reduction up to the age of 11 years.[154] The 1980 Report states: "These studies suggest unfavorable effects of maternal smoking during pregnancy on the child's long term growth, intellectual development, and behavioral characteristics."[155]

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[154] Id., Ch. 8 at 28. 1980 Surgeon General's Report on Women and Smoking at 237.

[155] 1980 Surgeon General's Report on Women and Smoking at 237.

G. Smoking and the Use of Birth Control Pills

The synergistic effect of smoking and the birth control pill makes the millions of women who use estrogen-containing oral contraceptives and also smoke cigarettes particularly subject to disability and death from cardiovascular diseases.[156] It is now well established that women who both smoke and use birth control pills are more likely to suffer heart attacks than would be expected by adding the separate risk factors for smoking and for contraceptive use. A woman who uses estrogen-containing birth control pills doubles the likelihood that she will suffer a myocardial infarction, or heart attack.[157] If she also smokes, her risk multiplies dramatically and she becomes ten times more likely to suffer a heart attack than a woman who neither smokes nor uses the pill.[158] Heavier smokers who use oral contraceptives face an even greater risk of heart attack, as do women in their mid-thirties and forties.[159]

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[156] 1979 Surgeon General's Report, Ch. 4 at 60-61.

Virtually all of the birth control pills prescribed in the United States contain estrogen. IMS National Prescription Audit (1978). The most recent and complete data available indicate that in 1976 over six million women who were married or had at one time been married used birth control pills. This total does not include those women who had never been married who used birth control pills and thus probably underestimates the number of women who use oral contraceptives. Vital Health Statistics of the National Center for Health Statistics, Department of Health, Education and Welfare, Advance data (No. 36, 1978 and No. 40, 1978).

[157] 1979 Surgeon General's Report, Ch. 4 at 60.

[158] 1979 Surgeon General's Report, Ch. 4 at 60.

[159] 1980 Surgeon General's Report Smoking and Women at 116-17. *See also*, Shapiro, *et al*, *Oral Contraceptive Use in Relation to Myocardial Infarction.*, The Lancet, 743-47 (1979).

Women who smoke and use oral contraceptives also appear to increase greatly their chances of suffering one type of stroke caus by a cerebral hemorrhage. The 1979 Surgeon General's Report cites recent study finding that oral contraceptive use by a woman who doe not smoke makes her about six times more likely to develop a subarachnoid hemorrhage than a woman who neither smokes, nor takes birth control pills.[160] In contrast, women who both take oral contraceptives and smoke cigarettes are about 20 times more likely suffer a subarachnoid hemorrhage than women who neither smoke nor take the pill.[161]

The normal incidence of heart attack and subarachnoid hemorrhag among the age group of women who ordinarily use oral contraceptives is relatively low, even with the enormous increase in risk caused b the combined use of birth control pills and cigarettes. As a result the total number of women who actually suffer from this disease is not of epidemic proportions. However, the problem is substantial enough that the Food and Drug Administration has required that the Patient Package Insert for all oral contraceptives prominently display the following statement:

> Cigarette smoking increases the risk of serious adverse effects on the heart and blood vessels from oral contraceptive use. This risk increases with age and with heavy smoking (15 or more cigarettes

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[160] 1979 Surgeon General's Report, Ch. 4 at 61.

[161] Id.

> per day) and is quite marked in women over 35 years of age. Women who use oral contraceptives should not smoke.[162]

As the FDA statement has concluded, women who smoke and use oral contraceptives greatly increase their risk of suffering cardiovascular diseases.

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[162] 21 C.F.R. § 310.51 (1980).

H. Smoking and Addiction

After reviewing evidence regarding the compulsive use, toxicity and adverse social consequences of cigarette smoking, a panel of experts convened by the National Institute on Drug Abuse ("NIDA") concluded that "...cigarette smoking behavior should be considered a form of addiction, and tobacco in the form of cigarettes, an addicting substance."[163] The experts recommended that policies toward cigarette smoking should be "...re-examined in light of the range of policy considerations which are presently considered germane to the classic forms of drug addiction such as addiction to the narcotics, sedatives, stimulants, or alcohol."[164] Recently, the National Advisory Council on Drug Abuse adopted a policy statement formally requesting the Department of Health and Human Services to recommend that Congress require that all cigarette packages include a warning that cigarettes are addictive.[165]

In addition to the addictive biological and pharmacological effects of cigarette smoke, psychological factors play an important role in maintaining cigarette use, as they do for addictions to other substances.[166] Nonetheless, most experts on the psychological aspects of smoking agree with the NIDA conclusion that most cigarette smokers are in some way physically dependent on cigarettes. However, despite clear evidence that some persons can become physically dependent on tobacco smoke,[167] scientists and physicians have not yet been able to demonstrate conclusively which component or components of the smoke make it so difficult for people who want to stop smoking to actually do so. At the present time, nicotine is thought to be the primary addicting element, but more research may uncover additional addicting components in cigarettes and define nicotine's role more clearly.[168]

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[163] National Institute on Drug Abuse of the U.S. Public Health Service, Final Report: Technical Review on Cigarette Smoking as an Addiction, ("NIDA") (1979) at 6.

The NIDA panel used the following definition of an addicting substance: "An addicting substance is one that has: (1) pharmacological properties leading to compulsive use; (2) a capability of producing organ and/or behavioral toxicity; and (3) a use pattern associated with adverse social consequences." Id., at 2.

[164] Id., at 6.

[165] National Advisory Council on Drug Abuse, Minutes of Meeting, May, 1980.

In evaluating the effect of nicotine many experts caution that tested nicotine levels of a cigarette may not accurately reflect the actual amount of nicotine taken in by a smoker.[169] For example, there is some evidence that smokers of low-yield cigarettes compensate for reduced nicotine by inhaling more deeply, puffing mor often, smoking closer to the cigarette butt, or by covering air hole with their lips or fingers.[170] If so, smokers may be receiving more nicotine, "tar" and other substances than the cigarettes' "tar' and nicotine levels would suggest. The concerns about compensation obviously extend beyond addiction to the effects on other diseases associated with smoking.

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[166] See generally 1979 Surgeon General's Report, Ch. 16 at 3-30.

[167] According to the NIDA Report, "Clear signs of withdrawal appear when heavy smokers abruptly quit, although there appears to be considerable variability in its manifestation. When a smoker stops smoking suddenly, he/she frequently shows a decrease in heart rate, sometimes in blood pressure, and a decrease in excreted epinephrine and nonepinephrine and its metabolities. Other endocrinological changes may also occur. Furthermore, there is a decrease in mean EEG frequency, an increase in appetite and weight, and an impairment in performance on psychomotor tasks and in concentration. Disturbances in arousal and sleep may occur; and anxiety, irritability, and aggression increase. Finally, there is an increase in craving for smoking which decreases with time." NIDA Report, Id. at 5.

[168] 1979 Surgeon General's Report, Ch. 15 at 7.

[169] This note of caution was echoed by both the Behavioral Science and the Pharmacology and Toxicity groups at the June 1980 Low Yield (Continued)

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169 (Footnote Continued)

Cigarette Conference of distinguished scientists and physicians sponsored by the Department of Health and Human Services, Office of Smoking and Health ("Low Yield Conference"). Both groups emphasize the need for further study of smoker compensation for reduced nicotine yield. Low Yield Conference Transcript, supra at 8, 28.

[170] Id.

I. Low "Tar" Cigarettes

Since the issuance of the 1964 Surgeon General's Report,[171] the average "tar" and nicotine content of cigarettes has declined. This decline was encouraged by the 1966 Public Health Service announcement that "the preponderance of scientific evidence strongly suggests that the lower the 'tar' and nicotine content of a cigarette, the less harmful [will] be the effect."[172] The F.T.C. also issued a policy statement taking this position.[173]

In the early 1970's, the cigarette companies began to promote aggressively low "tar" and nicotine cigarettes.[174] More recently, there has been a dramatic sales shift toward these cigarettes. In 1971, less than 4% of all cigarettes sold in the United States were low "tar".[175] By 1978 28% of the domestic cigarettes sold had 15.0 mgs. or less of "tar".[176] By 1979, low "tar" cigarettes accounted for slightly less than 41% of that year's sales.[177] Ultra-low "tar" cigarettes, which have 6.0 mg. or less "tar", have substantially lower sales, accounting for less than 6% of total sales in 1979.[178]

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[171] The 1964 Surgeon General's Report identified "tar" and nicotine as the major known harmful constituents of cigarette smoke. 1964 Surgeon General's Report at 62.

[172] Hearings before the Consumer Subcomm. of the Senate Comm. on Commerce, 90th Cong., 1st Sess. (1967). The Public Health Service always accompanied this statement with the caveat that the best way to prevent the hazards of smoking was not to smoke at all. Office of Smoking and Health, Department of Health and Human Services, Low Yield Conference, Transcript for June 9, 1980, at 9. The policy statement was written by Drs. E. Cuyler Hammond and Ernst Wyndor. Id.

[173] Id.

[174] As early as the 1950's, cigarette companies advertised that certain brands were lower in "tar" and nicotine and, by implication, less dangerous. In the absence of uniform testing procedures, it was impossible to make claims about "tar" and nicotine levels that could be substantiated and, in 1960, the FTC obtained the agreement of cigarette manufacturers to stop making claims about "tar" levels.

What is the health significance of this shift in smoking behavior? By Congressional directive the entire 1981 Report of the Surgeon General is devoted to this topic.[179] As then Secretary

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[175] The FTC's Annual Report to Congress on Cigarette Advertising for the Year 1978 at 15. The FTC formally defines a low "tar" cigarette as one that has 15.0 or less milligrams of "tar". As indicated in the table in fn. 178, the informal definition of "low tar" is changing.

[176] Id.

[177] F.T.C.'s Annual Report to Congress on Cigarette Advertising for the Year 1979 (forthcoming).

[178] Id. The table below shows the 1979 domestic market share for cigarettes of the "tar" levels indicated. Id.

Percent of Total Sales	
15mg & below	40.9%
12mg & below	30.5
9mg & below	10.6
6mg & below	5.8
3mg & below	2.7

[179] Office on Smoking and Health, U.S. Department of Health and Human Services, The Health Consequences of Smoking, The Changing Cigarette, A Report of the Surgeon General (January 12, 1981) ("1981 Surgeon General's Report").

Harris noted in transmitting the 1981 Report to Congress:

> In preparing this report, the scientists and scientific agencies of this Department have reviewed all current scientific evidence and have concluded that the search for less hazardous cigarettes has not yielded a product which can be considered "safe". The person who changes to a cigarette with lower measured yields may reduce certain hazards of smoking, but the benefits will be small compared to the benefits of quitting entirely.[180]

After viewing all of the evidence the Surgeon General concluded:

> 1. There is no safe cigarette and no safe level of consumption.
>
> 2. Smoking cigarettes with lower yields of "tar" and nicotine reduces the risk of lung cancer and, to some extent, improves the smoker's chance for longer life, provided there is no compensatory increase in the amount smoked. However, the benefits are minimal in comparison with giving up cigarettes entirely. The single most effective way to reduce hazards of smoking continues to be that of quitting entirely.
>
> 3. It is not clear what reductions in risk may occur in the case of diseases other than lung cancer. The evidence in the case of cardiovascular disease is too limited to warrant a conclusion, nor is there enough information on which to base a judgment in the case of chronic obstructive lung disease. In the case of smoking's effects on the fetus and newborn, there is no evidence that changing to a lower "tar" and nicotine cigarette has any effect at all on reducing risk.
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> * * *
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> 7. A final question is unresolved, whether the new cigarettes being produced today introduce new risks through their design, filtering mechanisms, tobacco ingredients, or additives. The chief concern is additives. The Public Health Service has been unable to assess the relative risks of cigarette additives

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[180] Id..

> because information was not available from manuturers as to what these additives are.[181]

The Surgeon General further continued:

> In evaluating the public health significance of reduced risk of lung cancer, it is important to recognize that the largest component of excess mortality caused by smoking is cardiovascular disease deaths. There is not sufficient evidence to conclude that use of lower "tar" and nicotine cigarettes causes any reduction in this burden. The same is true of the other major diseases caused by cigarette smoking, most notably chronic obstructive lung disease and adverse effects on pregnancy.[182]

Given the current state of scientific knowledge, it is, therefore, necessary to proceed cautiously before concluding whether low "tar" and nicotine cigarettes are significantly less dangerous than other cigarettes. The conclusion in the 1979 Surgeon General's Report that smoking is hazardous to health is based upon a review of some 30,000 published articles and eight large prospective epidemiological studies conducted over a period of thirty years.[183] Much less research has been done on the effect of lower "tar" and nicotine cigarettes. The lack of empirical evidence of the beneficial health effects of lower "tar" and nicotine cigarettes and the need for more research on this subject is a recurrent theme emphasized throughout the 1981 Surgeon General's Report.[184]

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[181] Id., at ii.

[182] Id., at ii.

[183] 1979 Surgeon General's Report, Ch. 7 at 5.

[184] 1981 Surgeon General's Report, Ch. 4 at 120-121, Ch. 5 at 139, 146-148, Ch. 6 at 157, 159.

Lack of empirical evidence is not the only reason for the need to proceed cautiously before concluding that lower "tar" and nicotine cigarettes are necessarily safer than those higher in "tar" and nicotine. Cigarette smoke contains over 2000 known compounds. In addition to "tar" and nicotine, cigarette smoke contains many other harmful substances, including such gases as carbon monoxide, hydrogen cyanide, nitrogen dioxide and volatile aromatic hydrocarbons.[185] The existence of these and other potentially harmful constituents in cigarette smoke increases the possibility that reducing the "tar" and nicotine levels without reducing the levels of these other agents will not eliminate some of the most serious health hazards of smoking.

For example, although scientists still are uncertain which agents cause heart disease, many suspect that nicotine and carbon monoxide are the critical compounds.[186] Scientists also suspect that the irritating gases in cigarette smoke may play a major role in causing chronic obstructive lung disease.[187] Although the medical evidence is not conclusive, carbon monoxide may be the most important factor in causing spontaneous abortion, still birth, neonatal death and low birthweight babies.[188] The role of numerous other possibly toxic substances in cigarette smoke, such as cadmium and cyanide, in altering fetal growth and development also needs to be studied further.[189] The evidence thus indicates that as a result of the presence of these other ingredients in cigarette smoke a reduction of "tar" and nicotine will not necessarily eliminate all or even most of the health hazards of smoking unless the levels of the other constituents are also reduced.

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[185] Id., 1979 Surgeon General's Report, Ch. 1 at 29-30; 1981 Surgeon General's Report, Ch. 2 at 33-34.

[186] 1979 Surgeon General's Report, Preface at xiii; 1981 Surgeon General's Report, Ch. 4 at 117-119.

[187] 1981 Surgeon General Report, Ch. 5 at 148-149.

[188] 1979 Surgeon General's Report, Ch. 8 at 57-67; 1981 Surgeon General's Report, Ch. 6 at 167-169.

There is also evidence that some of the benefits which might accrue from a reduction in "tar" and nicotine are to some unknown extent offset by two factors. First, many low "tar" cigarettes contain flavoring agents and additives to replace the flavor lost by reducing the "tar" levels.[190] The health effects of these additives and flavoring agents are not yet known and some may themselves be carcinogenic.[191]

Second, there is evidence that some smokers who switch to low "tar" cigarettes compensate for the reduced level of nicotine by a variety of methods that increase their intake of "tar" and nicotine to levels substantially above those indicated by machine testing.[192] Some smokers compensate by inhaling more often, inhaling deeper, smoking the cigarette closer to the filter, or simply smoking more cigarettes.[193] The construction of many of the low yield cigarettes also contributes to the intake of "tar" and nicotine above the levels obtained by the FTC testing apparatus. These cigarettes have a series of tiny holes around the middle of the filter. By covering these holes with one's finger or lips, a smoker increases the level of "tar" and nicotine inhaled.[194] Although the evidence suggesting that many smokers of low "tar" and nicotine cigarettes compensate for the reduced delivery of these constituents is not conclusive, it is also not possible to conclude at this time that compensation does not occur.

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[189] 1981 Surgeon General's Report, Ch. 6 at 167-169.

[190] 1979 Surgeon General's Report, Preface at xiii; 1981 Surgeon General's Report, Ch. 2 at 51-52, 55-56, 60.

[191] *Id*.

[192] Kozlowski, Frecker, Khouw, Pope, *The Misuse of "Less Hazardous" Cigarettes and its Detection: Hole Blocking of Ventilated Filters*. 70 American Journal of Public Health 1202-1203 (1980); 1979 Surgeon General's Report, Preface at xiii; 1981 Surgeon General's Report, Ch. 7 at 180-181.

Some experts believe that smokers of low "tar" and nicotine cigarettes find it easier to quit smoking altogether than do people who smoke higher "tar" and nicotine brands, although others cite evidence that low "tar" smokers do have difficulty quitting. If the former is true, switching to a low "tar" cigarette may be the first step toward stopping smoking entirely and would offer an undisputed health benefit to those persons who used them as a step toward quitting. However, there is also some evidence that the availability of low "tar" and nicotine cigarettes makes it easier for some people to *start* smoking or to rationalize not quitting. Based on data indicating that teenage boys and especially teenage girls tend to smoke low "tar" brands in greater proportion than the population at large, one researcher hypothesizes that teenagers and young adults, particularly girls, find it easier to experiment with and later become habituated to low "tar" cigarettes.[195]

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[193] 1979 Surgeon General's Report, Appendix at 20-21; 1981 Surgeon General's Report, Ch. 7 at 180-181.

[194] See Hoffman & Wynder, The Low Yield Cigarette, 70 American Journal of Public Health, 1144-46 (1980), in which the authors emphasize the need for sound scientific data on how actual "tar" and nicotine delivery relates to tested levels of "tar" and nicotine. The authors state that it is necessary to develop "low yield filter cigarettes that are less vulnerable to the smoker's manipulation of their actual smoke and nicotine deliveries." Id., at 1144.

If this is true, it could offset the benefit gained by those who use low tar cigarettes as a first step in quitting. It should be stressed that the evidence on the *use* of low "tar" cigarettes as a method of quitting and the evidence that these cigarettes make it easier for some people to start smoking or to rationalize not quitting is far from conclusive.

In conclusion, the findings of the 1981 Surgeon General's Report re-enforce the cautious position taken by the Surgeon General two years earlier:

> Until these scientific and behavioral issues are resolved, there can be no final assessment of the public health benefits of our present search for less hazardous cigarettes. The preponderance of scientific evidence continues, as in 1966, to suggest that cigarettes with lower "tar" and nicotine are less hazardous. It has become clear in the years since, however, that in presenting this information to the public three caveats are in order: Consumers should be

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[195] Harris, J., *Public Policy Issues in the Promotion of Less Hazardous Cigarettes* Banbury Report 3: A Safe Cigarette? (G. Gori & F. Bock, eds. 1980) at 333-36.

> advised to consider not only levels of "tar" and nicotine but also (when the information becomes available) levels of other tobacco smoke constituents, including carbon monoxide. They should be warned that, in shifting to a less hazardous cigarette, they may in fact increase their hazard if they begin smoking more cigarettes or inhaling more deeply. And most of all, they should be cautioned that even the lowest yield of cigarettes presents health hazards very much higher than would be encountered if they smoked no cigarettes at all, and that the single most effective way to reduce the hazards associated with smoking is to quit.[196]

Two strong proponents of low "tar" and nicotine cigarettes have reached a similar conclusion.

> Above all, we must not lose sight of the fact that cessation of smoking among adults and educational efforts to prevent onset of the habit among children remain the ultimate choices for eliminating the health risks associated with tobacco usage.[197]

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[196] 1979 Surgeon General's Report, Preface at xiv (emphasis added).

[197] Hoffman & Wynder, The Low Yield Cigarette, 70 American Journal of Public Health 11, 1144-46 (1980).

J. The Tobacco Industry's Position on the Health Effects of Smoking

Although the cigarette manufacturers no longer claim that cigarette smoking has affirmative health benefits, they continue to argue that it has not been proven that smoking has any detrimental health effects. The tobacco industry makes four general arguments in support of its position. They are: (1) Epidemiological evidence,[198] which establishes a correlation between smoking and the incidence of a disease, cannot be used to establish a causal link between smoking and that disease no matter how strong the epidemiological evidence. (2) Smoking cannot be identified as a cause of a particular disease without a full understanding of how that disease develops and the specific agent in cigarette smoke that leads to its development (disease etiology). (3) Unidentified individual (constitutional) differences between smokers and nonsmokers, and not smoking itself, explain why smokers suffer from certain diseases more often than nonsmokers. (4) All of the studies which have been used to establish a link between smoking and health are flawed in design or methodology.[199]

Central to the tobacco industry's position is the contention that epidemiological evidence never can be relied upon to demonstrate that smoking increases one's likelihood of developing certain diseases.[200] The industry contends that epidemiological evidence, which relies on establishing statistical correlations between the incidence of a given disease and the presence of a suspected disease-causing agent, cannot be used by itself to establish a cause and effect relationship. However, use of epidemiological evidence to help establish a causal link between a particular agent and a particular disease represents a well accepted practice among the medical profession. In 1964 the Surgeon General considered and rejected the argument that massive epidemiological evidence cannot be used to help establish causation:

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[198] See the explanation of epidemiology in Chapter I, fn. 23, supra.

[199] These arguments may be found in a review of Smoking and Health, The Tobacco Institute, 1979 at 37-41. ("Smoking and Health").

> Statistical methods cannot establish proof of a causal relationship in an association. The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability. To judge or evaluate the causal significance of the association between the attribute or agent and the disease, or effect upon health, a number of criteria must be utilized, no one of which is an all-sufficient basis for judgment. These criteria include:
>
> a) The consistency of the association
> b) The strength of the association
> c) The specificity of the association
> d) The temporal relationship of the association
> e) The coherence of the association[201]

The epidemiological evidence demonstrating the association between smoking and the diseases discussed in this report meet these criteria. The Surgeon General found that the association is consistent, strong, specific and independent of other identifiable risk factors. For example, every major epidemiological study has shown a strong and consistent association between smoking and lung cancer.[202] Smokers do not simply develop more cases of lung cancer. With all other variables held constant, the more cigarette they smoke, the more years they smoke or the younger they are when they start smoking, the more likely they are to develop lung cancer.[203] In addition, people who stop smoking reduce their risk of developing lung cancer.[204] This dose-response effect shown by epidemiological evidence based upon the number of cigarettes smoked, duration of smoking and age of initiation of smoking, supports the finding that there is a cause and effect relationship between smoking and lung cancer.[205] Similar dose-response relationships have also

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[200] *Smoking and Health, supra* at 41-42.

[201] 1964 Surgeon General's Report at 20.

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[202] 1979 Surgeon General's Report, Ch. 5 at 11.

[203] *Id.*, Ch. 5 at 31.

[204] *Id.*

[205] *Id.* In addition to its general argument that correlation does not establish causation, the Tobacco Institute alleges that one cannot conclude that cigarette smoking causes cancer because the following factors have not been considered critically: (1) diagnostic advances, (2) changes in reported frequencies of lung cancer cell types and (3) trends in cigarette consumption and lung cancer mortality. *Smoking and Health, supra* at 87.

Examining these factors does not alter the conclusion that smoking causes lung cancer. Diagnostic advances may explain why doctors are better able to identify lung cancer in all people, but they do not explain why smokers are far more likely than nonsmokers to develop lung cancer. Smoking has been linked to squamous cell carcinoma, which has been decreasing in incidence, as well as to adenocarcinoma, which has been increasing in incidence. In fact, smoking is related to the development of all four major cell-types o lung cancer in men and women. 1979 Surgeon General's Report, Ch. 5 at 23-24; 1980 Surgeon General's Report on Smoking and Women at 131.

Trends in cigarette consumption and lung cancer mortality also help establish, rather than disprove, that smoking causes lung (Continued been found between smoking and the incidence of coronary heart disease,[206] chronic obstructive lung disease,[207] perinatal death,[208] and reduced infant birth weight.[209]

In sum, the epidemiological evidence linking smoking to numerous diseases is strong and consistent. In many cases, it is corroborated by clinical and experimental evidence.[210]

The tobacco industry's second argument that scientists cannot establish causation until they fully understand the etiology of a disease is completely uncompelling. Scientists know that *something* about cigarette smoke causes chronic obstructive lung disease. For example, the Tobacco Institute argues that because scientists do not know exactly how smoking causes chronic obstructive lung disease, smoking cannot be called the major cause of the disease.[211] However, smokers develop the disease far more often than nonsmokers;

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205 (Footnote Continued)

cancer. For example, the lung cancer death rate among women is now rising steeply, paralleling the increase in the number of women who smoke that began after World II. 1980 Surgeon General's Report on Smoking and Women at 7.

[206] 1979 Surgeon General's Report, Ch. 4 at 22-25.

[207] *Id.*, Ch. 6 at 10.

[208] *Id.*, Ch. 8 at 47.

[209] *Id.*, Ch. 8 at 13.

[210] *Id.*, Ch. 4 at 40, 45-46; Ch. 5 at 29-31, 34-35, 41-42, 44, 47, 51-53; Ch. 6 at 23-24; Ch. 8 at 52-65.

[211] *Smoking and Health*, *supra* at 134.

those who smoke more or longer get the disease more often; and those who quit smoking eliminate the acceleration of symptoms.[212] Just as physicians knew that eating limes or other citrus fruits prevented scurvy long before they knew which compound in citrus fruits actually prevented the disease or exactly how it did so, it is not necessary to know which component of cigarette smoke causes chronic obstructive lung disease, or exactly how the component affects the lung, to conclude that smoking causes emphysema and chronic bronchitis.[213] Research funded by the tobacco companies reached the same basic conclusion. Even though the etiology of chronic obstructive lung disease had not been established, the AMA-ERF Committee stated: "The Committee believes that the bulk of research sponsored by this project supports the contention that *cigarette smoking plays an important role in the development of chronic obstructive pulmonary diseases* ..."[214]

The tobacco industry's third argument contends that the increased incidence of various diseases among smokers might be a result of individual characteristics of the people who smoke, and not of smoking itself.[215] This "constitutional" argument alleges that the types of people who smoke are more likely to suffer various diseases, not because they smoke, but because of underlying constitutional differences between smokers and nonsmokers. However, proponents of the constitutional argument have been unable to identify any characteristic other than smoking that accounts for the increased risk of disease faced by smokers. More important, the evidence that people who quit smoking reduce their risk of lung cancer,[216] heart attack,[217] chronic bronchitis and emphysema,[218] and low birthweight babies,[219] is fundamentally inconsistent with the constitutional differences hypothesis. If one is constitutionally more likely to suffer certain diseases, the fact that she/he quits smoking would not reduce the person's chances of developing the diseases.

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[212] 1979 Surgeon General's Report, Ch. 6 at 9-10.

[213] Similarly, physicians were also able to prevent smallpox long before they knew exactly what the causative agent was. Low Yield Conference, Transcript for June 9, 1980 at 44; 1979 Surgeon General's Report, Ch. 5 at 31.

[214] *Tobacco and Health*, American Medical Association Education and Research Fund, 1978 at xiv (emphasis added).

[215] *Tobacco and Health, supra* at 35, 92-93; *See also*, Seltzer, *Smoking and Coronary Heart Disease: What Are We To Believe?* 100 American Heart Journal 275 (1980).

The tobacco industry's fourth argument concerns the design and methodology of the studies relied upon to show the health hazards of smoking. Perhaps the most important evidence about smoking and health comes from the studies that show that smokers have higher mortality rates than nonsmokers. The tobacco industry, through the

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[216] 1979 Surgeon General's Report, Ch. 5 at 24-25.

[217] Id., Ch. 4 at 34.

[218] Id., Ch. 6 at 22-23.

[219] Id., Ch. 8 at 12.

Tobacco Institute, has attacked the design or methodology of the major studies on which these findings are based.[220] The 1979 Surgeon General's Report examined the criticized studies and found that they were sound in design and methodology.[221] While it is necessary to examine carefully the design and methodology of any study, there have been numerous massive epidemiological studies of the health effects of smoking which have all reached the same conclusion: cigarette smoking is hazardous to health. Therefore, given the extraordinarily large number of epidemiological studies on this subject employing a wide variety of designs and methodologies which all reach the same basic conclusions, the likelihood that they are all so flawed that their conclusions are erroneous is miniscule.

While all that will eventually be known about the specific health consequences of cigarette smoking is not known now, the evidence that smoking is dangerous has grown continuously for more than the last three decades and can now only be described as overwhelming.[222] The Surgeon General, the World Health Organization, the American Medical Association, the American Heart Association, the American Cancer Society, the American Lung Association and members of the insurance industry, all recognize that smoking is dangerous.[223] Even studies funded by the tobacco companies conclude that smoking increases the risk of certain diseases.[224] The four basic arguments asserted by the tobacco industry in defense of its position that smoking has not been proven to have any serious adverse health effects do not stand up to rigorous analysis.

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[220] Smoking and Health, supra at 37-41.

[221] 1979 Surgeon General's Report, Ch. 2, fn. 25 at 43. Actuarial evidence gathered by the insurance industry corroborates the conclusion reached in the 1964 and 1979 Surgeon General's reports that smokers have higher death rates than nonsmokers.

[222] Id., Preface at vii.

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[223] See Chapter I, Section I. A., supra.

[224] Tobacco and Health, American Medical Association Education and Research Fund, 1978 at xiv.

CHAPTER II: CIGARETTE ADVERTISING*

I. INTRODUCTION

This chapter will examine the type, amount and content of cigarette advertising in recent years. Cigarette advertising continues to be pervasive in American society. Although it has been prohibited from the broadcast media for the past decade, cigarette advertising has shifted to the print and outdoor advertising media to such an extent that it is a dominant force in both areas.

The purposes of the presentation of the information in this chapter relate only to concern that cigarette advertisements which do not effectively disclose the health hazards of smoking may be deceptive. This chapter reviews recent cigarette advertising to assist in an evaluation of the effectiveness of the current warning and to understand the overall advertising context in which the current warning appears and in which it must compete to effectively communicate its health message. In contrast to the current warning, cigarette advertisements present information about smoking in a highly effective manner. They communicate their message about smoking in a variety of attention-getting, frequently changing formats. The ads are rich in thematic imagery associating smoking with, among other things, outdoor activities, athletics, individualism and achievement. They are frequently filled with rugged, vigorous, attractive, healthy-looking people living energetic lives full of success and athletic achievement, free from any health hazards.

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* In addition to the information in this chapter, the Commission relied upon information in confidential marketing plans and a confidential report on the results of focus groups. This information has been deleted from the version of the report released to the public.

Not only are most cigarette advertisements filled with this rich, thematic imagery, many may even more strongly divert or distract attention away from the health consequences of smoking by portraying smoking as compatible with or, at least, as associated with a wide range of rigorous athletic or other strenuous activities. It is possible that these ads make it more difficult for the health warning to be effective and may further increase the possibility of deception. The impact on the efficacy of the current warning of the advertisements in which it appears was aptly summarized in an Advertising Age article describing a Christmas advertisement for Marlboro:

> A valley of snow holds a log farmhouse under a blanket of white. Smoke rises in the still air from the chimney. A lone cowboy rides his horse through the untouched virgin snow, dragging a Christmas tree by a rope....It's hard to imagine a more evocative American image, even though the

> white boxed cancer warning in the right hand corner has stained the snow yellow. The reflective pleasure of tobacco pervades the ad. It unifies the desire for a perfect Christmas with the experience of smoking. The Surgeon General has no chance against this.[1]

II. THE AMOUNT OF CIGARETTE ADVERTISING IN THE UNITED STATES

Cigarettes are the most heavily advertised product in America.[2] It has been estimated that the six major cigarette companies spent one billion dollars in 1980 to sell their product.[3] This figure is several hundred times greater than the amount government spends on public service announcements on smoking hazards.[4] The National Commission on Smoking and Public Policy reported in 1978 that the tobacco industry spent more on advertising cigarettes in one day than the (then) National Clearinghouse on Smoking and Health, the government's primary agency working in this area, spent in one year.[5]

Cigarette advertising expenditures have grown substantially over the last decade and continue to grow. Preliminary figures show that expenditures for 1980 ran 26% ahead of the 1979 rate.[6] The real growth in cigarette advertising between 1967 and 1979 exceeded 50%,[7] despite the passage of the Public Health Cigarette Smoking Act, which barred cigarette advertising from the broadcast media after January 1, 1971.[8]

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[1] Advertising Age, December 25, 1978, at 18.

[2] Leading National Advertisers, Jan.-Dec. 1979.

[3] Marketing and Media Decisions, October, 1980, at 176. The six major companies are: American Tobacco, Brown & Williamson, Liggett & Myers, Lorillard, Philip Morris, and R.J. Reynolds.

[4] Blum, Commentary: Medicine v. Madison Avenue, 243 Journal of the American Medical Association 739 (1980).

[5] Report of the National Commission on Smoking and Public Policy to the Board of Directors, American Cancer Society, Inc., A National Dilemma: Cigarette Smoking or the Health of Americans (1978) at 9.

In 1979, two of the top five advertisers in magazines in the United States were cigarette companies.[9] R.J. Reynolds was the largest single advertiser in magazines, and its expenditures included 25 million dollars spent on the promotion of just one of its many brands, Winston.[10] Philip Morris was also among the top five magazine advertisers in this country, spending close to 60 million dollars.[11] Lorillard, Brown & Williamson and American Tobacco, although spending less than the two industry leaders, were all among the top users of magazine advertising.[12]

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[6] Marketing and Media Decisions, October, 1980, at 176. The figures are $121,000,000 for 1st quarter 1980, $95,982,000 for 1st quarter 1979.

[7] Federal Trade Commission Bureau of Economics estimate. In deriving this estimate, the Bureau has controlled for the effects of inflation.

[8] 15 U.S.C. § 1331 et seq. (1970).

[9] Advertising Age, September 11, 1980, at 48.

[10] Marketing and Media Decisions, October, 1980, at 190.

[11] Id. at 176.

[12] Id.; Advertising Age, Sept. 11, 1980, at 48.

In 1979, cigarettes also continued to be the product most heavily advertised in newspapers.[13] Lorillard's Kent was the most advertised single brand, with expenditures of over 30 million dollars.[14] Merit, manufactured by Philip Morris, was the second most advertised brand at over 26 million dollars.[15] Newspaper advertising for all cigarettes during the year increased by 28.3% over the 1978 figures.[16]

The tobacco industry dominates outdoor advertising even more than it does magazines and newspapers. The top five outdoor advertisers in 1979 were the five largest cigarette companies.[17] Almost half of all billboards in the United States advertise cigarettes. Moreover, R.J. Reynolds, the largest manufacturer of cigarettes, decided recently to increase substantially its billboard advertising to become the number one presence in all outdoor advertising.[18] The large and still increasing use of outdoor cigarette advertisements is especially important because the current health warning is probably least effective on a billboard. (See, infra, Appendix D.)

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[13] *Marketing and Media Decisions*, June, 1980, at 103-04.

[14] *Id.*; *Marketing and Media Decisions*, October, 1980, at 176.

[15] *Marketing and Media Decisions*, October, 1980, at 176.

[16] *Marketing and Media Decisions*, June, 1980, at 103-04.

[17] *Advertising Age*, Sept. 11, 1980, at 78.

[18] *Advertising Age*, Sept. 22, 1980, at 110; *Adweek*, Sept. 15, 1980, at 1, 58.

Transit advertising, the volume of which is not measured nationally, is also an increasingly important part of the cigarette companies' advertising program. The amount of money spent by the cigarette industry on this form of advertising increased from 5 to 22 million dollars between 1970 and 1978. The increase will be even greater in 1980 and 1981 because R.J. Reynolds is also increasing its transit budget significantly.[19]

Another form of promotion not widely used before the broadcast ban, but which has become important in recent years, is the sponsoring of music and sporting events by the cigarette companies. Notable examples of this form of advertising include the Virginia Slims tennis circuit and the Kool Newport Jazz Festival in New York. The use of discount coupons and free cigarette samples given out on street corners is another promotional technique on the rise. One source estimates that cigarette industry expenditures on this form of advertising have increased 1000 percent over the past five years.[20] For example, Brown & Williamson, in promoting a new low "tar" brand, Barclay, has been distributing free cartons of cigarettes. If this promotional technique is successful, it could cost the company up to $80 million dollars.[21]

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[19] Id.

[20] Blum, Commentary: Medicine v. Madison Avenue, 243 Journal of the American Medical Association 739 (1980).

The rise in the amount of money being spent to advertise all cigarettes over the last decade also has been accompanied by a shift in the percentage of advertising expenditures allocated to the low "tar" and nicotine market. The low "tar" market, defined as cigarettes with less than 15 mg. "tar", has increased from 2% in 1967, to 27.5% in 1978,[22] to an estimated 50% in 1980.[23] Advertising expenditures for low "tar" cigarettes have paralleled this increase, rising from 5.5% of cigarette advertising expenditures in 1967 to 48% in 1978.[24] The percentage of cigarette advertising dollars spent on low "tar" cigarette ads in 1979 was 66.9%.[25] Philip Morris is expected to spend $50 million this year alone on its new low "tar" brand, Cambridge,[26] and Brown & Williamson's competitors estimate that it will spend up to $150 million to promote Barclay.[27]

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[21] Fortune, Nov. 17, 1980, at 121.

[22] Table 11, FTC's Annual Report to Congress on Cigarette Advertising for the Year 1978.

[23] Marketing and Media Decisions, October, 1980, at 176.

[24] Table 11, FTC's Annual Report to Congress on Cigarette Advertising for the Year 1978.

[25] Preliminary figures for the FTC's Annual Report to Congress on Cigarette Advertising for the Year 1979.

[26] Marketing and Media Decisions, October, 1980, at 176.

[27] Fortune, Nov. 17, 1980, at 121.

III. RECENT TRENDS IN CIGARETTE ADVERTISING

The Commission's Statement of Basis and Purpose for the 1964 Cigarette Rule noted that two themes predominated in then current cigarette advertising: portrayals of the desirability of smoking and assurances about the safety of cigarettes or the relative safety of particular brands.[28]

In the ensuing sixteen years, there has been little change in the character of cigarette advertising.[29] Cigarette ads have continued to attempt to allay anxieties about the hazards of smoking and to associate smoking with good health, youthful vigor, social and professional success, and other attractive ideas, individuals and activities that are both worthy of emulation and distant from concerns relating to health. Most cigarette advertisements express at least one of these themes. Often more than one such theme can be found in a single advertisement. Thus, the cigarette is portrayed as an integral part of youth, happiness, attractiveness, personal success and an active, vigorous, strenuous lifestyle.

A Benson & Hedges campaign, for example, features young men and women engged in various athletic and outdoor activities. The slogan, "B&H, I like your style," captions pictures of a couple with tennis rackets, a couple riding a bicycle for two, a woman with a surfboard, a woman climbing the rigging of a sailboat, a man assisting a woman who has fallen on the ski slopes, a woman driving a racing car, a man and woman roller skating, a woman carrying logs through the snow, a man riding a unicycle, a woman on snowshoes, a man and woman on horseback, a woman golfer, and a woman cross-country skier. In nearly all of these situations, involving healthy young people engaged in rigorous, athletic activity, the models are holding cigarettes. Previous Benson & Hedges campaigns have portrayed men and women sailing, fishing, surfing, skating, skiing, riding, golfing, playing baseball, and biking.

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[28] 1964 Cigarette Rule, supra, 20 Fed. Reg. at 8341.

[29] See Appendix A for a variety of cigarette ads illustrating themes discussed in this section.

Newport ads feature couples in situations emphasizing vigor, fun and novelty. These poses include a woman on horseback in water holding hands with a man floating on raft; a couple on a beach with a trombone; a man and woman leaping over a fire hydrant; a couple on a toboggan with a dog; a woman turning a somersault in front of admirers on the beach; a man shooting a bow and arrow with a woman nestled in his arms; and a couple sliding down a slide into a pool of water. The ads exclaim: "Alive with pleasure" or "Revive your taste!"

Previous Newport campaigns have featured young people engaged in all manner of outdoor and athletic activities - football, frisbee, basketball, skiing, snorkeling, kite-flying, swimming, snowmobiling, and tobogganing. One Newport model rides a bicycle with a woman perched on the handlebars and a cigarette in his hand. Ads for many cigarette brands feature models in tennis outfits with rackets in hand. Advertisements for Belair cigarettes portray various kinds of beach activities.

In a related vein, a number of cigarette campaigns associate smoking with strenuous outdoor activity, and a clean, outdoor environment. One ad for Old Gold filters shows a young man with a backpack smoking a cigarette on a mountain ledge overlooking an expansive green valley. Most Salem ads feature a lake or stream, surrounded by green foliage, with snow-capped mountains in the background. The copy of one such ad reads: "Light mountain breezes. Clear rippling waters. And country fresh Salem menthol." Menthol Kool likewise advertises the taste of "menthol mist" against a backdrop of waterfalls or ocean spray.

Themes of vitality, ruggedness, strength and individualism are closely related to the images of athletic prowess described above. For example, a Camel Filters ad, declaring that "some men taste it all," features a handsome and rugged-looking young man in a life jacket, lighting up next to his raft which is beached near white water rapids, with two attractive young women in shorts looking on nearby. Another Camel Filters ad campaign presents "the Turk," a handsome young man, often surrounded by beautiful women, who is described as "one of a kind," a man who "is at home in a world few men ever see." The famous Marlboro man, galloping across the range on horseback or swinging a lasso, is well known for his rugged macho image. Various attractive young models in Winston advertisements claim: "I don't smoke to be like everybody else."

The original, unsuccessful campaign for low "tar" Real cigarettes, which featured a tobacco leaf and long copy discussing the brand's characteristics, was replaced by ads picturing a dune buggy racing across the sand or a jeep being driven through rapids. Inserts show the rugged-looking, powerfully-built drivers smoking cigarettes. The image projected is that of adventurous, active young smokers.

Many cigarette advertisements feature strikingly attractive and healthy young models, such as Camel's Turk and the handsome, bare-chested man and beautiful woman who have appeared in Winston ads. The use of such models associates smoking with physical attractiveness and youth. Some Winston ads imply that smoking is an activity that is begun before one "grows up." One such ad features a woman who appears to be in her twenties stating that: "Winston wasn't my first cigarette. I learned about smoking by trying different cigarettes...Winston may not be where you start. But when your taste grows up, Winston is for real." Another such ad pictures a male lumberjack who declares: "When your taste grows up, Winston out-tastes them all."

An ad for Silva Thins features a slender woman in a low-cut evening dress. The text reads: "Long, lean and low with lots of style." A Max ad, picturing an attractive model dressed in high fashion, urges: "Wear a Max today. Long, lean, all-white, tasteful." In More ads, on the other hand, black and white males and females assert that they are "More satisfied" because of the "style" they get from a "long, slim, brown cigarette."

A number of campaigns imply that smoking a particular brand solves the health problem or at least minimizes the risks. Vantage employs signed testimonials by smokers who claim to have considered the risks of smoking and decided not to quit smoking, but rather to switch to Vantage. The headlines say: "Smoking. Here's what I'm doing about it," or "With Vantage, I don't have as many problems with smoking."

Advertisements for brands such as Tareyton and Parliament imply that their special filters minimize the risks of smoking. For example, a recent Parliament ad claims that the cigarette's recessed filter prevents the smoker's lips from touching the "tar" that "builds up on the tip." Ads for 120mm cigarettes, such as Max and Saratoga, claim that the smoker can "enjoy smoking longer without smoking more."

An earlier True cigarette ad featured a serious-looking young woman in tennis garb, standing on a tennis court with a cigarette in hand, saying: "Considering all I'd heard, I decided to either quit or smoke True. I smoke True." This ad incorrectly implies that when the alternatives of quitting smoking or smoking a low "tar" cigarette are weighed, the low "tar" cigarette is the healthier option.[30]

All of the above examples of cigarette advertising evidence the continuing viability of the FTC's 1964 observation that:

> ...cigarette advertising is replete with descriptions of satisfactions to be derived from smoking. These descriptions are both explicit and varied. Their constant repetition in advertising which reaches vast numbers of Americans of all ages must be viewed as significantly contributing to the portrayal of the desirability of smoking.[31]

Thus, the dominant themes of cigarette advertising are that smoking is associated with youthful vigor, good health, good looks and personal, social and professional acceptance and success, and that it is compatible with a wide range of athletic and healthful activities. One theme is conspicuously absent from all cigarette ads. Although these ads contain the required general warning, they make no mention of the numerous and specific adverse health consequences of using the advertised product.[32]

[Confidential Information Omitted]

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[30] For a discussion of the medical evidence with regard to low "tar" and nicotine cigarettes, see Ch. I, Sec. II, supra.

[31] 1964 Cigarette Rule, supra, 20 Fed. Reg. at 8342.

[32] To the best of our knowledge, cigarette companies never have provided health information, other than the required warning and "tar" and nicotine figures, in their advertisements.

Chapter III. CONSUMER KNOWLEDGE OF THE HEALTH * HAZARDS OF SMOKING

I. INTRODUCTION AND OVERVIEW

This chapter presents the existing empirical evidence concerning consumer knowledge about the health hazards of smoking. From this data it is possible to determine the extent to which consumers are aware of or lack knowledge about the health consequences of smoking.

A. Data Relied Upon

The following chapter includes the staff's analysis of: (1) the already existing survey data of the public's knowledge of the risks of smoking, (2) two nationally projectable surveys commissioned by the F.T.C. staff designed to measure consumer knowlege in this area and (3) several studies commissioned by the staff not designed to directly measure consumer knowledge, but which nevertheless provide insight into it, such as the Walker Study and the Burke Focus Group, discussed below. In all, staff evaluated more than a dozen recent studies relevant to consumer knowledge. The most important studies are briefly described below:

Gallup Study:

This 1978 nationally projectable face-to-face opinion poll of 1500 persons surveyed smoking attitudes and behavior, and included questions concerning beliefs about the health hazards of smoking. The Gallup Opinion Index, Smoking in America, Public Attitudes and Behavior, Report No. 155, June 1978.

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* In addition to the information in this Chapter, the Commission relied upon information in two confidential nationally projectable surveys. This information has been deleted from the version of the report released to the public.

The Shor Study:

This 1980 study assessed 307 college students' perceptions and beliefs about the effects of environmental smoke and the health hazards to smokers. Shor et. al., *Beliefs of Smoking and Nonsmoking College Students About the Effects of Environmental Tobacco Smoke and Related Issues*, 10 J. Drug Education 274 (1980).

1978 Roper Report:

This nationally projectable survey of people's attitudes about smoking was conducted for the Tobacco Institute, and submitted to the FTC in response to a subpoena. The survey was conducted via face-to-face interviewing with 2,511 subjects. Part of the report, summarizing the results, was appended to the Commission's 1978 Report to Congress. The Roper Organization, Study of Public Attitudes Toward Cigarette Smoking and the Tobacco Industry in 1978.

Chilton Survey:

This 1979 nationally projectable telephone survey, conducted for the FTC, questioned 1,211 teenagers aged 13-18 and 407 adults aged 29-31 about their beliefs concerning the health effects of smoking. Chilton Research Services, A Survey of Adolescent and Adult Attitudes, Values Behavior, Intentions and Knowledge Related to Cigarette Smoking, 1979.

Walker Study:

This study of 805 persons, conducted for the FTC in 1980, was designed to test the ability of several new health warnings to make people think about the health hazards related to smoking. In addition, the warnings were tested for their understandability. The study provides insight into consumer knowledge through the relative ranking of proposed warnings, and more directly, through analysis of verbatim comments of subjects in the study explaining why they preferred some warnings over others. Walker Research, Health Warning Concepts Study, 1980.

Burke Study:

A 1980 focus group interview study conducted for the FTC to assess responses and attitudes towards proposed health warnings' formats, Burke also provides insight into consumer knowledge of health hazards. A focus group interview consists of small discussion groups moderated by trained professionals. The moderator then records her impressions of the discussion in an "analysis" which constitutes the results of the study. While no statistical inference may be drawn from a focus group study, it can often provide richer and more "human" insight concerning people's beliefs and attitudes than can survey data. Burke Exploratory Print Focus Group, 1980.

HEW Teenage Smoking Study:

This study, conducted in 1979, interviewed by telephone 1,318 teens, ages 12-18, and included questions reflecting teen knowledge of the health hazards of smoking. Teenage Smoking: Immediate and Long Term Patterns, National Institute of Education, 1979.

[Confidential Information Omitted]

1980 Roper Study:

A face-to-face nationally projectable survey conducted by Roper for the FTC. Two-thousand respondents were tested for knowledge about health hazards of smoking. The field dates were November 15-22, 1980.

In reviewing this data it is important to remember that it is extremely difficult to test for consumer knowledge empirically. To some extent, test results may vary depending on the methodology employed in a particular study and the phraseology of the particular questions. General, conservative sounding statements have been found to be more likely to generate agreement than bold, direct sounding ones, even if both are true and have similar meanings.[1] Similarly, aided recall (multiple choice) questions may yield higher "correct" responses than unaided questions. In addition, there is always some element of error in any survey attributable to numerous factors, including survey design, mistaken responses, compilation errors, and the like. It may also be possible that there is a small portion of those unaware of the hazards of smoking who are unreachable on the issue: that is, no matter how much information is presented to them, they will not understand that smoking is dangerous to them.[2] In short, consumer data and the methodology used always must be carefully scrutinized.

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[1] Thus, Chilton found that 90% of the population believed that smoking has "been found to be associated" with heart attack. [Confidential Information Omitted]

The studies discussed in this report represent a substantial body of data concerning public awareness of the hazards of cigarette smoking. The surveys cited polled more than 8,000 respondents, and asked hundreds of questions on virtually all of the many important health consequences associated with smoking. Thus, in reviewing this Chapter, it is not necesary to rely on any single study, the results of any single question or the data as it relates to consumer knowledge about just any one particular disease or health risk of smoking. Therefore, the probability of error or bias in the overall results is substantially reduced. The studies also employ a wide variety of accepted survey methodologies, which insures that the Commission need not rely on any single methodology or question-type in assessing consumer knowledge.

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[2] It is possible that some small number will continue to respond incorrectly to questions about the hazard of smoking no matter how much information is available to them. For example, some smokers may respond incorrectly because they do not want to believe or admit that they are endangering their own health. While it is not unlikely that some fall into this category, their existence or precise number is speculative and unsupported by empirical evidence. In any case, given both the large number of people who the data indicate do not know about many of the most serious health effects of smoking and the variations in the percentage of people who respond correctly depending on the health information being surveyed, this theory does not account for the major gaps in knowledge revealed by the survey data discussed in this chapter.

Moreover, a review of the results indicates that the data are consistent between studies, and, according to the 1980 Roper study of consumer knowledge, consistent along sex, race, age, socioeconomic, and geographic variables. These consistencies further suggest that the data are reliable and valid. Finally, to permit the reader to draw his or her own conclusions from the data, staff has presented the evidence in the following section in considerable detail, generally including the question asked as well as the results. In sum, the combined results of all the studies discussed in this section represent the most comprehensive evidence available of overall consumer knowledge, beliefs, and awareness of the health hazards of smoking.

F. THE RELEVANCE OF SPECIFIC HEALTH INFORMATION

Staff has proceeded in this investigation on the assumption that most people are generally aware that smoking is hazardous. To a large extent, this assumption is supported by the data. Thus, although it is clear that not every American knows smoking is hazardous--indeed, Gallup found that 24% of heavy smokers do not know or believe it is hazardous--most Americans are aware of this basic fact. However, the data also indicate that many Americans are not aware of any more specific information about smoking and health.

There are several reasons why this more specific health information is relevant to consumers. First, knowledge of more specific information is relevant to an understanding of how dangerous smoking is; i.e., what is the nature and general extent of the health risk of smoking. Second, knowledge of more specific information is relevant to consumers' awareness of whether the general health risks of smoking have any personal application to themselves or whether they are among those groups of people who may be uniquely vulnerable to these health hazards. Third, numerous studies demonstrate that consumers have a more difficult time remembering general or abstract information, are more likely to consider it personally irrelevant, and are less likely to consider health information at all without more specific information.

(1) Knowledge of more information about the health risks of smoking is relevant to an understanding of the general risk involved, i.e., how dangerous smoking is. Many consumers who have heard that the Surgeon General has determined that smoking is dangerous do not generally know, for example, (a) what diseases are smoking related, (b) what is the generally increased likelihood of suffering from these diseases as the result of smoking, or (c) what effect these diseases have on the likelihood of suffering a premature death.

Typically, in other situations in which consumers make decisions which affect their health, they want and are provided more specific information. For example, patients consenting to surgery are told more than the general fact that the surgery may somehow be dangerous to their health. The patient is told what the potential harmful consequences of the surgical procedure are, how likely it is that the patient will suffer from these consequences and how serious these consequences are likely to be, including how likely it is that the patient might die as a result of the surgery. Similarly, a woman who sees an advertisement for birth control pills or some other birth control device is told more than the fact that in some cases these pills or devices have been found to be hazardous to health. She also is informed about the specific potential health consequences. How likely is she to suffer from them? Is she within one of the categories of women who is particularly susceptible and, therefore, more likely to suffer these health consequences? She is also informed about how serious and permanent are these health consequences. In the smoking decision, as well as in these examples, some knowledge of the specific health consequences and the increased risk of the consequences from smoking is relevant to an understanding of how dangerous smoking really is.

Consumers have also indicated that they do want more information about the specific health hazards of smoking. One out of every four persons polled by Roper in the 1978 study felt they "need to know more" about smoking and health.[3] Roper also found that the majority of consumers favored a stronger, more specific warning. Eighty-two percent of the sample said they favored health warnings for cigarettes,[4] and 61% favored a new warning detailing the specific hazards associated with smoking over the current warning.[5]

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[3] 1978 Roper, q. 68.

[4] 1978 Roper, q. 9.

[5] 1978 Roper, q. 25. The statement tested was "Warning: Cigarette smoking is dangerous to health and may cause death from cancer, coronary heart disease, chronic bronchitis, pulmonary emphysema and other diseases." This warning was proposed by the FTC in the Annual (Continued)

(2) Knowledge of more information about the health hazards of smoking is relevant to consumers' awareness of whether the health risks associated with smoking have any personal application to themselves or whether they are among those people who are particularly vulnerable to these health hazards. For example, some consumers incorrectly believe that while smoking may be hazardous to others, it is not hazardous to themselves because of the particular type of cigarette they smoke or the amount they smoke. Other consumers are at special risk if they smoke, but are unaware of this fact. Included, among others, in the former group, are consumers who incorrectly believe that smoking only a few cigarettes each day is not harmful, consumers who incorrectly believe that low "tar" and nicotine cigarettes are safe, and consumers who incorrectly believe, despite the evidence indicating that smoking may be addictive, smoking is safe as long as you quit before it becomes a habit. Of particular concern in this group are teenagers who are unaware of the potentially addictive nature of cigarette smoking, and who, therefore, may be tempted to "experiment" with smoking. Included in the latter group are pregnant women, women who take birth control pills, and persons with pre-existing medical conditions which are aggravated by smoking.

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[5] (Footnote Continued)

Report to Congress on Cigarette Advertising for the Year 1978. Staff tested a similar warning in the Walker study.

(3) It is more difficult for consumers to remember and consider health information without more specific, concrete information about the health hazards of smoking.[6] Numerous studies indicate that people rely on concrete information much more than on abstract information in making judgments and decisions. Indeed, it has been shown that there is a dramatic overreliance on concrete information accompanied by a corresponding underreliance on abstact information in making decisions. Concrete information is also better remembered than abstract information. Pictures are better remembered than words, and concrete words are better remembered than abstract words. For example, the word "smoke" creates an easily remembered mental picture, whereas the word "hazardous" does not. Similarly, it has been shown that consumers are more likely to remember and consider information which they perceive as having personal implications and relevance to themselves. Specific, concrete information is much more likely to be perceived as personally relevant than abstract, general information. There is a large difference between being aware of the statement that "smoking is dangerous" and believing that "my smoking will injure me." The former is seen as an abstract statement which is difficult to interpret in terms of one's personal experiences. Thus, general knowledge that smoking is hazardous to health is much less likely to be considered personally relevant and material by consumers than knowledge of the important health hazards of smoking.

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[6] For a more detailed discussion of and citation to appropriate authorities for the facts mentioned in this paragraph, See Ch. IV, Sec. II.

Based upon an analysis of all the data reviewed in its entirety, staff has concluded that there appear to be such significant gaps in consumer knowledge that a substantial portion of the population does not know how dangerous smoking is, or whether the dangers of smoking apply to them, and, therefore, does not have an understanding of the health hazards of smoking. More specifically, the data suggest that (a) a large number of consumers do not know what diseases are related to smoking; (b) a substantial number seriously misunderstand and underestimate the extent of the increased risk of suffering these health problems as the result of smoking; (c) many seriously underestimate or are unaware of the severity of these health consequences and the increased risk of premature death from these illnesses; and (d) a significant number of consumers incorrectly do not believe that these health consequences have any personal relevance to themselves.

II. OVERALL RISK AND REDUCTION IN LIFE EXPECTANCY

It has been almost 17 years since the Surgeon General first determined that cigarette smoking is hazardous to health. For 15 years, warnings to that effect have appeared on cigarette packages and for almost ten years, warnings have also appeared on every cigarette ad. Yet, survey data indicate that a substantial portion of people remain unaware of the serious overall health risk associated with smoking.

The data, projected nationwide, indicate that some Americans still do not even know that smoking is hazardous to health. In 1978, Gallup asked respondents whether they believed smoking was harmful to health. Although 10% of the sample did not know that smoking had any harmful effects,[7] 17% of all smokers did not know that smoking is harmful to health, and nearly one out of every four - 24% -heavy smokers did not know this fact.[8] In the 1978 Roper study, 9% of the total sample, 13% of all smokers, and 20% of two pack or more a day smokers did not know that smoking was hazardous.[9] Given that there are approximately 54 million smokers in this country, these data suggest that millions of Americans who smoke still do not know that smoking is harmful to their health.

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[7] Gallup Opinion Index, June, 1978 at 20, 21; 7% "No", 3% "Don't know."

[8] All smokers: 13% "No", 4% "Don't know"; Heavier smokers (one or more packs a day): 19% "No", 5% "Don't know."

[9] The Roper Organization, Study of Public Attitudes Toward Cigarette Smoking and the Tobacco Industry in 1978 ("1978 Roper"), q. 16. Overall: 5% "Smoking isn't hazardous", 4% "Don't know"; Smokers: 8% "Not hazardous", 5% "Don't know"; 2 pack or more a day smokers: 13% "Not hazardous, 7% "Don't know."

More significantly, the data reveal that many consumers do not know that smoking significantly decreases a person's life expectancy, a critical fact for consumers to know in order to understand how dangerous smoking is to them. A thirty year old smoker will shorten his life on the average by between six and eight years if he smokes a pack a day.[10] However, among those polled in the 1980 Roper study, 30% of the population and 41% of the smokers did not know that a typical thirty year old male shortened his life expectancy at all by smoking.[11] Among those who did know that smoking reduces one's life expectancy, many seriously underestimated the degree to which this is true. On the average, nonsmokers underestimated this loss in life expectancy by about 2 years, while smokers underestimated it by more than four years.[12]

Similar results were obtained when respondents were asked if they thought a forty-year old man increased his risk of dying within the next year by smoking a pack a day. By smoking a pack a day, a man doubles his risk[13] of dying within the next year, but 44% of the sample, and 54% of smokers, did not know smoking increased at all a forty year old's risk of death within the next year.[14]

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[10] 1979 Surgeon General's Report, Chapter 2 at 12.

[11] 1980 Roper, q. 30. Overall: 22% "Don't know if it's true", 6% "think it's not true," 2% "know it's not true." Smokers: 27% "Don't know if it's true", 10% "think it's not true", 4% "know it's not true."

[12] 1980 Roper, q. 32. Based on median score, counting those who answered "know it's not true" or "think it's not true" to screening questions as "zero", and excluding those who answered "don't know if it's true" to the screening question, as well as those who did not answer either question.

These findings are generally consistent with the findings of earlier surveys. In the 1978 Roper study, Half of the people polled, and two-thirds of smokers, did not think that smoking made "a great deal of difference" in life expectancy. [Confidential Information Omitted] This evidence suggests, thus, that substantial numbers of people do not know that smoking reduces their life expectancy. The data also indicate that most people seriously underestimate the number of years one's life expectancy is reduced due to smoking, and the extent to which the risk of early death is increased by smoking.

Another way to assess knowledge of overall risk is to ask whether consumers know that smokers are more likely to get sick than nonsmokers. The responses to such a question put by Roper also revealed that overall 34% of the total sample, and 50% of the smokers, thought the statement, "smokers have more of certain illness than non-smokers," was not true, or only "possibly" true.[16] These data are important because they suggest that many still falsely believe that there is a substantial controversy about the basic fact that smoking is in any way hazardous to health.

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[13] See tables in 1979 Surgeon General's Report, Ch.2, at 16-20.

[14] Roper, q. 30. Overall: 28% "Don't know if it's true"; 11% "Think it's not true"; 5% "Know it's not true." Smokers: 27% "Don't know if it's true", 18% "Think it's not true," 9% "Know it's not true."

When those who did know that there was some increased risk were asked to quantify the risk, three times as many people chose the lowest alternative - "less than 15%" - than chose the correct answer, "100% (doubles the chance)", and the median was "less than 15%."

[15] [Confidential Information Omitted]

The 1978 Roper study also indicates that a great many consumers share a common misperception: they believe, incorrectly, that only heavy smoking is dangerous. Medical evidence clearly indicates that smoking only a few cigarettes a day can be harmful. Eight major studies, using age adjusted mortality ratios, showed that smokers of

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[16] 1978 Roper, q. 14.

> IT'S BEEN SAID THAT SMOKERS HAVE MORE OF CERTAIN ILLNESSES THAN NON-SMOKERS. WOULD YOU SAY THIS IS DEFINITELY TRUE, PROBABLY TRUE, POSSIBLY TRUE, OR NOT TRUE?

	Total	Total Smokers
Definitely	33	17
Probably	29	28
Possibly	23	30
Not True	11	20
Don't know/No answer	4	5

Moreover, of those who did feel that smokers have more illnesses, the vast majority did not feel that smoking was the major cause. Sixty-eight percent of the sample, and 85% of smokers, who felt that smokers had more of certain illnesses believed either that smoking was one of many causes, not a cause, or didn't know the reason for the excess illness. Only 32% of the sample, and 15% of smokers, felt that smoking was the major cause of excess illness in smokers. Computed from 1978 Roper, q. 15.

only 1-9 cigarettes per day increased their risk of premature death by 20 to 45%.[17] However, nearly a third of the total sample, and two-fifths of smokers, believed that "only heavy smoking is hazardous."[18] When asked what they meant by heavy, all but a handful of respondents said at least a pack a day.[19] Fifty percent of "light" smokers[20] felt that only heavy smoking is hazardous, suggesting that many people incorrectly believe that *their* smoking habit is not dangerous, because they do not consider themselves "heavy" smokers. Similarly, the 1980 Roper study indicates that about one out of three smokers does not know that smoking without inhaling into one's lungs is also dangerous.[21] Each of these facts suggests that substantial numbers of smoking consumers falsely believe smoking is not hazardous to *them personally*.

A recent study of 307 college students at the University of New Hampshire (Shor study)[22] revealed another way consumers underestimate the overall risk from cigarette smoking. The students were presented with the following medically accurate statement: "In industrialized nations, cigarette smoking is the greatest single cause of excess morbidity and mortality from lung and other cancers, from heart attacks, and also from emphysema in both men and women." Nearly half of the college-educated sample disbelieved the statement, or were uncertain.[23]

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[17] 1979 Surgeon General's Report, Ch. 2 at 16, Table 4.

[18] 1978 Roper, q. 16/17. 31% total, 40% smokers. *See also*, *Teenage Smoking: Immediate and Long-Term Patterns*, National Institute of Education (1979). Twenty-four percent of teenage boys and 21% of teenage girls agreed with the statement, "There's nothing wrong with smoking cigarettes if you don't smoke too many." Among teen smokers, 43% of boys and 26% of the girls agreed with this false statement.

[19] Computed from 1978 Roper, q. 16/17.

[20] Roper defined a light smoker as one who smokes 1 to 9 cigarettes per day. 1978 Roper Report and Summary at 42.

[21] 1980 Roper, q. 30. 21% "Don't know if it's true", 8% "Think it's not true", 3% "Know it's not true."

[22] Shor, *et al.*, *Beliefs of Smoking and Nonsmoking College Students*

(Continued)

In sum, the data concerning the overall health risk of smoking indicate that while most consumers are aware of the general fact that smoking is hazardous, many consumers apparently do not know how dangerous smoking is and seriously underestimate the overall health risk to them from smoking. This conclusion is further supported by evidence discussed below.[24]

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[22] (Footnote Continued)

About the Effects of Environmental Tobacco Smoke and Related Issues, 10 J. Drug Education 263 ("Shor") (1980).

[23] Shor, q. 21. Overall, 34% were "Neutral or uncertain," 15% "Disbelieved" or "Strongly disbelieved." It is possible that some of the uncertainty expressed in response to this question may be due to the technical wording of the question.

[24] A 1979 study commissioned by the FTC and conducted by Chilton Research Services, A Survey of Adolescent and Adult Attitudes, Values, Behavior, Intentions and Knowledge Related to Cigarette Smoking ("Chilton") (1979), also suggests that people greatly underestimate the relative risk of death from smoking. Respondents were asked which caused the most deaths during the last year - traffic accidents, fires, drug overdose, or cigarette smoking. The correct answer is, by far, cigarette smoking, but more than twice as many people chose traffic accidents than chose smoking. Among teens, smoking came in third, as more teens thought drug overdose was the number one killer rather than smoking. Of smokers, only 14% of teens and 17% of adults knew that cigarette smoking was the number one killer

(Continued)

III. CANCER

Studies consistently indicate that cigarette smoking increases the risk for cancer of the lung, larynx, mouth, esophagus, bladder, kidney, and pancreas, leading the Surgeon General to conclude that smoking has been "implicated as a significant cause"[25] for all of these cancers. The survey data indicate that consumers evidence greater knowledge about the smoking-cancer link than about the other health hazards of smoking. However, the data also indicate that some, and perhaps many, people remain unaware of the cancer risk. In addition, the data suggest that substantial numbers of consumers who may know that smoking is related to cancer seriously underestimate the extent to which smoking increases their cancer risk and the extent to which a smoker increases his/her risk of death from cancer.

While most consumers know that smoking is linked to cancer, a number of people still do not believe the link between smoking and any type of cancer. [Confidential Information Omitted]

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[24] (Footnote Continued)

among the items listed. Chilton, q. 23. See, also Chilton, q. 17, where nearly 90% of the sample underestimated the number of annual deaths caused by smoking, and more people chose the lowest alternative - 10,000 - than the correct answer (based on conservative estimates) of 300,000. See, also Slovic, Fischoff & Lichtenstein, Rating the Risks, 21 Environment 14, 20 (1979) where the authors report that the health risk from smoking was consistently underrated when subjects were asked to quantify various health risks.

[25] 1979 Surgeon General's Report, Ch. 5 at 9.

In addition, substantial numbers of people also don't believe that smoking causes various specific cancers.

A. Lung Cancer

Medical experts agree that cigarette smoking is the major cause of lung cancer cases in both men and women.[27] In fact, it has been estimated that cigarette smoking accounts for about 85% of the lung cancer in this country.[28] Despite the evidence on this point,[29] there continues to be a substantial gap in public knowledge of the link between smoking and lung cancer. The data also indicate that smokers are more likely to have incorrect beliefs about the relationship between smoking and lung cancer than are nonsmokers.

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[26] [Confidential Information Omitted]

[27] 1979 Surgeon General's Report, Ch. 5 at 31.

[28] See, e.g., Holbrook, Tobacco Smoking, in Harrison's Principles of Internal Medicine, 960 (9th Ed. K. Isselbacher, 1980), noting that "[a]n estimated 80 percent of lung cancer cases are attributable to smoking, and are preventable." The Surgeon General estimated that out of 92,400 lung cancer deaths in 1978, about 80,000, or 88% of them were attributable to smoking. 1979 Surgeon General's Report, Secretary's Forward at ii; Ch. 5 at 10. See, also, Levin, M.L., Statement before the Committee on Commerce, U.S Senate, 89th Congress, First Session, Serial 89-5; March 23, 1965, pp. 144-148, estimating that 83.5% of all lung cancer deaths are attributable to smoking.

[29] 1979 Surgeon General's Report, Ch. 1 at 16.

According to the Gallup Opinion Index, June, 1978, 19% of the opulation do not believe that smoking causes lung cancer.[30] allup found a larger knowledge gap among smokers, and the largest ap among heavier smokers, whose risk for lung cancer is the greatest. Among all smokers, 28% did not believe smoking caused lung cancer while among heavier smokers,[31] nearly one-third -- 31% -- did not believe or know about the link.[32] [Confidential Information Omitted] Projected nationwide, these data suggest that tens of millions of Americans, both smokers and non-smokers, do not know that cigarette smoking causes lung cancer.

An even larger proportion do not appreciate the magnitude of the increased risk of lung cancer associated with smoking. One important fact relevant to understanding this increased risk is that smoking causes most cases of lung cancer. In fact, smoking accounts for about 85% of lung cancer cases. The 1980 Roper study found that 43% of the population, and 49% of smokers, don't know that smoking causes

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[30] Gallup Opinion Index at 22. Respondents were asked, "Do you think cigarette smoking is or is not one of the causes of lung cancer?" Eleven percent of the sample answered that smoking is not one of the causes, while another 8% said they didn't know whether it was or wasn't.

[31] Those who smoked one pack a day or more.

[32] Total smokers: 19% "Not a cause", 8% "Don't know"; Heavy smokers: 23% "Not a cause," 8% "Don't know."

[33] [Confidential Information Omitted]

most cases of lung cancer, and 22% of the population, 27% of smokers, don't know that smoking even causes *many* cases of lung cancer.[34] In the Chilton study, respondents were asked, "What percent of lung cancer cases are caused by cigarette smoking?" They were presented with four choices - 10%, 35%, 50%, and - the correct answer - 85%. The results indicated that the *majority* of all persons underestimated, and that more smokers than nonsmokers underestimated, the percent of lung cancer cases caused by smoking. Fifty-six percent of teens and 54% of adults underestimated the percent of lung cancer cases caused by smoking, and 12% of teens and 19% of adults answered "don't know." Among smokers, 62% of the teenagers and 59% of the adults underestimated. Many people *greatly* underestimated the percent of lung cancer cases attributable to smoking: 27% of teenagers (30% smokers) and 28% of young adults (34% smokers) thought the correct answer was either 10 or 35%.[35]

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[34] 1980 Roper, q. 29. In this question, respondents were asked to indicate their beliefs about smoking and certain health problems, including lung cancer. They were given four choices: smoking "Does not cause the problem"; "Causes some but not many cases"; "Causes many but not most cases"; "Causes most cases." For lung cancer, 21% responded "Many but not most", 14% "Some but not many", 3% "Does not cause," 5% "Don't know." Similarly, the 1980 Roper study found that 21% of the sample, and 31% of smokers, do not know that smoking is *by far* the greatest cause of lung cancer. 1980 Roper, q. 30. When presented with that statement, 13% responded "Don't know if it's true," 6% "Think it's not true," 2% "Know it's not true." Smokers, 17% "Don't know if it's true", 11% "Think it's not true," 3% "Know it's not true."

[35] Chilton, q. 20.

Since many people greatly underestimate the percent of lung cancer cases attributable to smoking, it is also likely that many people are unaware that smoking is the major cause of lung cancer. In the 1980 Roper study, 25% of all respondents failed to mention smoking when asked to name the number one cause of lung cancer.[36] In the 1978 Roper poll conducted for the tobacco industry, respondents were asked whether they agreed with the following false statement: "Air pollution is the major cause of lung cancer, not cigarette smoking." Twenty-two percent of the total sample agreed with this statement; 31% of the smokers polled agreed. In addition, 28% of the total sample and 29% of the smokers answered "don't know." In other words, responses to this question indicated that 50% of the total population, and 60% of smokers, do not realize that cigarette smoking is the major cause of lung cancer.[37]

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[36] 1980 Roper, a. 27.

[37] 1978 Roper, q. 18h. The difference between the 1978 and 1980 results might be explained by the fact that persons answering the open-ended question in the 1980 study were focusing on the words "lung cancer" rather than "number one cause" in the question. Thus, those who believed smoking caused lung cancer, whether or not they knew it was the number one cause, responded "smoking." The 25% who did not respond "smoking" is consistent with the data discussed above, which indicate that approximately that percent of the population do not know that smoking causes lung cancer. Results to this question do suggest that consumer knowledge about lung cancer and smoking is not "soft" - rather, no matter how you test it, about three-fourths of the population know that smoking causes lung cancer. The 1980 Roper study suggests knowledge about all types of cancer is relatively "hard" - i.e., results between aided and unaided (open-ended) recall questions are fairly consistent - in marked contrast to consumer knowledge concerning heart attack and pregnancy, where knowledge appeared much greater in response to multiple choice questions than to open-ended ones.

A cigarette smoker is about ten times more likely to develop lung cancer than a non-smoker. The 1980 Roper study tested consumer awareness of this fact in two ways. First, it asked consumers if they knew the specific fact that smokers are ten times more likely to die of lung cancer. Twenty-three percent of the population, and 39% of smokers, did not know this fact.[38] Some of this lack of knowledge, of course, is due to the use of a specific figure -- ten times -- in the question rather than real consumer misunderstanding. However, even when the question was phrased as a general statement, "Smokers are many times more likely to develop lung cancer than non-smokers," 16% of the total sample, and 25% of the smokers, did not know it was true.[39] This evidence further indicates that many consumers underestimate their increased risk of lung cancer if they smoke.[40]

In addition to the large number of consumers who still do not know that smoking causes lung cancer, and who do not know the magnitude of the increased risk of lung cancer for smokers, many people do not appreciate the severity of lung cancer. Seventy percent of lung cancer patients die within one year after diagnosis, and only 10% live for five years after diagnosis.[41] Of all those who get lung cancer, 95% eventually die from it.[42]

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[38] 1980 Roper, q. 30. Total: 15% "Don't know if it's true," 6% "Think it's not true," 2% "Know it's not true." Smokers, 22% "Don't know if it's true," 12% "Think it's not true," 5% "Know it's not true."

[39] Total: 11% "Don't know if it's true," 4% "Think it's not true," 1% "Know it's not true." Smokers: 17% "Don't know if it's true", 6% "Think it's not true," 2% "Know it's not true."

[40] Focus group research conducted for the staff by Burke Marketing confirmed this view. Burke concluded that although "[t]he greatest danger perceived for the smoker appears to be lung cancer...these persons did not have very definite ideas of how great the danger is. They seemed unsure of how much greater the probability for contracting lung cancer was for the smoker than for the non-smoker." Exploratory Print Focus Groups, January, 1980, Burke Marketing Research Inc.

In the Chilton study, respondents were asked "Out of every one hundred people who get lung cancer, how many die from it?"[43] In choosing between possible answers of 25%, 45%, 75%, and 95% (the correct answer), 80% of teenagers and 75% of adults underestimated the risk of death from lung cancer, while another 9% of teens and 14% of adults said they "didn't know." A large proportion of the sample greatly underestimated the correct percent: approximately one-half[44] of those questioned believed that either 25% or 45% was the correct answer.

B. Other Cancers

The data also suggest that significant numbers of people do not know that smoking causes other cancers, including cancer of the esophagus, mouth, larynx, and bladder. Smoking is a major cause of cancer of the esophagus, larynx, and mouth, and causes most cases of each of these deadly cancers.[45] In the 1980 Roper Study, however, 32% of the population, and 39% of smokers, did not know that smoking is a major cause of cancer of the mouth, larnyx, and esophagus.[46]

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[41] 1979 Surgeon General's Report, Ch. 5 at 31.

[42] Id.

[43] Chilton, q. 24.

[44] Fifty-one percent teens, 47% adults.

The Gallup Poll found that 21%[47] of the population did not know that smoking causes cancer of the esophagus. More smokers did not know about the relationship than did non-smokers: 27% of all smokers, and 30%[48] of one-pack a day or more smokers either thought that smoking did not cause throat cancer, or had no opinion.

A smaller, but still significant, if projected nationally, proportion of people are not aware of the link between smoking and cancer of the mouth. Chilton found that 18% of teens and 13% of adults did not believe that cancer of the mouth was associated with cigarette smoking.[49] A much larger percentage of the population do not know that smoking causes most cases of oral cancer. The 1980 Roper study found that 66% of the total population, and 70% of smokers, do not know that smoking causes most cases of cancer of the mouth. Moreover, 40% of the population, and 44% of smokers, did not even know that smoking causes many cases of oral cancer.[50]

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[45] See Ch. I, Sec. II, *supra*.

[46] 1980 Roper, q. 30. Total: 23% "Don't know if it's true", 6% "Think it's not true", 3% "Know it's not true"; Smokers: 27% "Don't know if it's true," 8% "Think it's not true", 4% "Know it's not true,"

[47] Ten percent answered "No, is not a cause"; 11% said they had no opinion. Gallup Opinion Index at 24.

[48] All smokers: 17% "No, is not a cause"; 10% "No opinion;" heavier smokers: 23% "No,....", 7% "No opinion."

[49] Chilton, q. 41c. It is worth emphasizing that Chilton asked respondents if they thought cigarette smoking "has been found to be associated" with cancer of the mouth. Since 13% of adults and 18% of teens did not know that smoking has even been found to be associated with oral cancer, it seems reasonable to assume that a larger proportion of the population is unaware that smoking *causes* cancer of the mouth. Indeed, the respondents who did not believe that smoking "has been found to be associated" with oral cancer presumably are not aware that there is *any* evidence linking smoking and oral cancer. Conversely, they need not personally be convinced that there is a (Continued)

Similar patterns were apparent when the 1980 Roper study tested for consumer knowledge of the relationship between smoking and cancer of the larynx. Sixty-two percent of the population, and 65% of smokers, did not know that smoking causes most cases of cancer of the larynx; 36% of the sample, and 38% of smokers, did not even know that smoking causes many cases of this disease.[51]

Studies indicate that, on the average, cigarette smokers are about twice as likely to die from cancer of the bladder as non-

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[49] (Footnote Continued)

real link between smoking and oral cancer to "agree" with the statement, as long as they believe at least one study "found" such an association. In other words, the phraseology used in Chilton, "found to be associated," is likely to generate a very high percent agreement, and probably overestimates the percent of people who understand the relationship between smoking and oral cancer. The same observations hold true for the identically worded questions in Chilton concerning heart disease, bronchitis, and emphysema, 42(b)(c) and (e).

[50] 1980 Roper, q. 29. Total: 26% "Causes many but not most," 24% "Causes some but not many," 7% "Does not cause," 9% "Don't know." Smokers: 24% "Causes many but not most," 31% "Causes some but not many," 8% "Does not cause," 7% "Don't know".

[51] 1980 Roper, q. 29. Total: 26% "Many but not most," 23% "Some but not many," 5% "Does not cause," 8% "Don't know." Smokers: 27% "Many but not most," 25% "Some but not many," 5% "Does not cause," 8% "Don't know."

smokers.[52] These studies have led the Surgeon General to conclude that bladder cancer is among those cancers caused by smoking. Yet, the overwhelming *majority* - 75% of adults and 62% of teens - of respondents in Chilton did not believe that cigarette smoking was associated with bladder cancer.[53]

In sum, these data suggest that while most people are generally aware that there is some relationship between smoking and cancer, substantial numbers of consumers do not know the extent to which smoking increases their risk of cancer or the extent to which smokers increase their risk of death from cancer. The data, therefore, suggest that a substantial portion of consumers do not have an understanding of their increased risk of cancer if they smoke.

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[52] 1979 Surgeon General's Report, Ch. 5 at 45.

[53] When asked, "Has cancer of the bladder been found to be associated with cigarette smoking?", only 38% of teens and 25% of adults answered "Yes" to the question, and 14% of teens and 26% of the adults said they didn't know. Chilton, q. 41(b).

IV. HEART DISEASE

Although mortality ratios are particularly high among cigarette smokers for diseases such as lung cancer and chronic obstructive lung disease, heart disease is the chief contributor to the excess mortality among cigarette smokers.[54] Smokers are, on the average, almost twice as likely to die from heart attack as are non-smokers.[55] Given the strength, consistency, predictive ability, and other characteristics of the association between smoking and heart disease, the Surgeon General has concluded that "smoking is causally related to coronary heart disease in the common-sense of that idea and for purposes of preventive medicine."[56] On the basis of these studies, smoking has been identified as one of the three major causes of coronary heart disease.[57]

However, according to survey data, many people remain uninformed about the relationship between smoking and heart disease. In the 1978 Gallup study, about one out of three - 32% - did not know that smoking caused heart disease.[58] Among smokers, 37%[59] did not know about the relationship; and, among heavier smokers (pack or more a day) 40%[60] did not know about the causal link. If projected nationally, these data suggest that over 50 million American adults, and over 20 million smokers, do not know that smoking causes heart disease.

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[54] 1979 Surgeon General's Report, Ch. 1 at 12.

[55] See, e.g., Hammond and Horn, 1958, who report a risk factor for all smokers of 1.70 (non-smokers=1.00); Doyle et al, 1964, report a risk factor for all smokers of 2.40. 1979 Surgeon General's Report, Ch. 4 at 22.

[56] 1979 Surgeon General's Report, Ch. 4 at 66.

[57] 1979 Surgeon General's Report, Ch. 4 at 21.

[58] Gallup Opinion Index at 25. Respondents were asked if they thought that cigarette smoking "is or is not" one of the causes of heart disease. Seventeen percent answered "is not", 15% answered Don't know."

[59] 25% "Is not," 12% "Don't Know."

Comparable gaps in consumer knowledge concerning the relationship between smoking and heart disease are evident in [Confidential Information Omitted] the Chilton study commissioned by the FTC staff, and the 1980 Roper study. [Confidential Information Omitted] While smoking doubles a person's heart attack risk, the 1980 Roper study found that 25% of the population, and 31% of smokers, did not know that smoking greatly increased their risk of heart attack.[62]

In Chilton, respondents were asked whether they thought the following correct statement was true or false: "Cigarette smoking is a major cause of heart disease." Twenty-seven percent of teens and 40% of adults responded either "false" or "don't know."[63] Once again, the gap in knowledge was greater among smokers: 33% of teenage smokers and 45% of adult smokers answered "false" or "don't know."[64] The 1980 Roper study found that 53% of the population and 58% of smokers do not know that smoking causes *many* cases of heart attack, and 20% of the population, 22% of smokers, do not even know that it causes *some* cases.[65]

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[60] 29% "Is not," 11% "Don't know."

[61] [Confidential Information Omitted]

[62] 1980 Roper, q. 30. Total: 17% "Don't know if it's true," 7% "Think it's not true," 1% "Know it's not true"; Smokers: 17% "Don't know if it's true," 12% "Think it's not true," 2% "Don't know if it's true."

In sum, the data support the conclusion reached by Burke Marketing Research, Inc., on the basis of focus group interviews, that, in terms of consumer knowledge, "[h]eart and circulatory problems are apparently not strongly associated with smoking yet."[66] The data suggesting that tens of millions of Americans are unaware of the leading cause of smoking-related death is consistent between several studies employing varied research methods and represents one of the major findings of this report concerning consumer knowledge.

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[63] Teens: 24% "False," 3% "Don't know;" Adults: 30% "False," 10% "Don't know." Chilton, q. 52.

[64] Teens: 32% "False", 1% "Don't know;" Adults: 33% "False," 12% "Don't know."

Chilton also asked respondents whether heart disease has "been found to be associated with cigarette smoking." About 10% of respondents would not agree even with this conservative statement concerning the relationship of smoking and heart disease.

[65] 1980 Roper, q. 29. Total: 33% "Some but not many", 12% "Does not cause", 8% "Don't know. Smokers: 36% "Some but not many", 14% "Does not cause", 8% "Don't know.

Similar findings, in a smaller study, were reported by Koslowsky & Croog, *Perception of the Etiology of Illness: Causal Attributions in a Heart Patient Population*, 47 Perceptual and Motor Skills 475-78 (1978). Three hundred forty-five heart patients were presented with 16 perceived causes that are common explanations for heart disease. Respondents were asked whether they believed each listed cause was a "very important", "important", or "not important" cause of heart attack. Forty-one percent of the sample said either that cigarette smoking was not an important cause of heart attack, or answered "Don't know."

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[66] Burke Exploratory Print Focus Groups (1980) at 1. Answers to an open-ended question in the 1980 Roper study also suggest that consumer knowledge of the heart attack-smoking relationship is "soft" - i.e., many consumers "know" about the relationship only in the context of aided recall (multiple choice) questions. About two-thirds of the sample did not respond "smoking" when asked to name all the causes they could think of for heart attack. 1980 Roper, q. 28. As only about one-third of the population doesn't know smoking causes heart attack when measured for aided recall, the data suggest about one-third of the sample know smoking causes heart attack with a relatively high level of awareness, one-third know it, but only with a low and perhaps ineffective level of awareness, and one-third don't know that smoking causes heart attack at any level of awareness. It is legitimate to ask whether consumers with a low-level of awareness, as discussed above, have an understanding of the risk of heart attack from smoking.

V. CHRONIC OBSTRUCTIVE LUNG DISEASE

Smoking is the single most important cause of chronic obstructive lung disease (COLD).[67] Smokers suffer a greatly increased risk for all types of COLD, including chronic bronchitis and emphysema.[68] The evidence indicates that smoking causes about 70% of all cases of chronic obstructive lung disease.

In Chilton, 18% of teens and 13% of adults did not know that chronic bronchitis "has been found to be associated" with smoking.[69] Twenty percent of teens and 9% of adults did not believe the statement for emphysema.[70]

While the majority of people know that smoking is related to these diseases, significant percentages don't know the magnitude of the increased risk. According to the 1980 Roper study, 59% of the population, and 63% of smokers, do not know that smoking causes most cases of emphysema, while 34%, and 39% of smokers, do not know smoking causes many cases.[71] For chronic bronchitis, consumer knowledge was even lower.[72]

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[67] 1979 Surgeon General's Report, Ch. 6 at 7.

[68] Id.

[69] Chilton, q. 42(b). Teens: 11% "No", 7% "Don't know"; Adults: 9% "No", 4% "Don't know". [Confidential Information Omitted]

[70] Chilton, q. 42(c). Teens: 12% "No", 8% "Don't know"; Adults: 5% "No" 4% "Don't know".

Experts estimate that about 30% of smokers in this country will suffer from chronic bronchitis. When this fact was presented as a proposed warning in the Walker research, many people tested expressed surprise, and appeared impressed. For example, one respondent said the warning "presents a startling fact";[73] another said the 30% figure "catches your attention".[74] Many other respondents made comments suggesting that they were both unaware of and impressed by this fact.[75] In sum, the data suggest many people do not know the extent to which smoking increases their risk of chronic obstructive lung disease.

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[71] 1980 Roper, q. 29. Total: 26% "Many but not most", 19% "Some but not many", 7% "Not a cause", 8% "Don't know". Smokers: 25% "Many but not most", 24% "Some but not many," 8% "Not a cause", 7% "Don't know".

[72] 1980 Roper, q. 29. Eighty-two percent of the population did not know that smoking causes most cases of chronic bronchitis, and 85% of smokers did not know it. Fifty-five percent, 60% of smokers, did not know it caused many cases. As bronchitis and emphysema are clinically indistinguishable, however, this greater lack of knowledge for chronic bronchitis may not be very significant.

[73] Subject #0520.

[74] Subject #0643.

[75] E.g., subject #0560, 0086, 0145, 0295.

In the 1980 Roper study, q. 32, subjects were presented with the statement "At least 30% of smokers will suffer from chronic bronchitis." Eleven percent of the sample and 17% of smokers said they knew or thought this was not true, while another 36% total, 37% smokers, said they did not know whether it was true.

Responses to a question in Chilton further support the view that many people are not aware of the extent of danger of lung disease from cigarette smoking. The question asked whether respondents believed the following false statement: "Air pollution is more unhealthy for your lungs than smoking cigarettes." Chilton, q. 54. (Continued)

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[75] (Footnote Continued)

Although the Surgeon General has determined cigarette smoking is "by far more important in producing respiratory disease" than air pollution, about one-quarter of all those asked, and one-third of the smokers asked, thought the above statement was true. Among adult smokers, fully 45% responded either "true" or "don't know" to this question, suggesting a significant level of misperception among those most likely to be suffering from respiratory disease.

VI. PREGNANCY AND HEALTH

A woman who smokes during pregnancy risks her own health, and that of her unborn child. Studies show that maternal smoking during pregnancy significantly increases the risk of miscarriage, stillbirth, and neonatal death, and leads to significantly smaller average birth weight.[76] However, a large percentage of people, particularly among those who smoke, are not aware of the serious consequences of maternal smoking during pregnancy.

In the 1980 Roper study, respondents were presented with the statement: "If a woman smokes during pregnancy, she significantly increases her risk of losing the baby before or during birth." Nearly one-half of the women polled -- 47% -- did not know of this fact.[77] In addition, the 1980 Roper study suggests that many of those who "know" this fact for purposes of an aided recall question have a relatively low level of awareness of the fact. When asked to name as many causes as they could think of for miscarriage and stillbirth, only 12% of the women polled volunteered "smoking."

A series of questions to test consumer knowledge in this area was also included in Chilton. First, respondents were asked whether maternal smoking during pregnancy had any effect on the baby. Next, those respondents who did believe smoking had an effect were asked specific questions about the relationship between smoking and miscarriage and stillbirth.

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[76] 1980 Surgeon General's Report on Smoking and Women at 224.

[77] 1980 Roper, q. 30. 29% "Don't know if it's true," 13% "Think it's not true," 5% "Know it's not true."

The results indicate that a small -- but significant if projected nationwide -- percent of the sample believed that maternal smoking during pregnancy had absolutely no effect on the baby: Ten percent of the total sample responded "No" or "Don't Know" when asked if maternal smoking had any effect on the fetus.[78]

When tested for knowledge of the specific relationships between smoking and miscarriage and stillbirth, consumers evidenced much larger knowledge gaps. In Chilton, 31% of all teenagers and 38% of all adults did not know or did not believe that a woman is more likely to have a miscarriage if she smokes during the last five months of pregnancy.[79] As an additional 10% of the sample did not think that maternal smoking had any effect on the baby (and thus were not asked this question on miscarriage), Chilton indicates that 41% of teens and 48% of adults do not know about the risk of miscarriage from maternal smoking. The knowledge gap among smokers was even greater: 52% of teenage smokers and 58% of adult smokers evidenced a lack of knowledge of this health consequence.[80]

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[78] Overall: 6% "No", 4% "Don't know". Chilton, q. 39. Smokers were more likely to doubt that smoking had any effect, and considerably more likely to believe that it didn't. Ten percent of teenage smokers and 9% of adults smokers answered "No" to the question, compared to 5% of non-smokers in each group. Twelve percent of teens smokers and 13% of adult smokers answered "No" or "Don't know" to the question, compared to 10% of teenaged nonsmokers and 9% of adult nonsmokers.

[79] Chilton, q. 40(a). Teens: 24% "No", 7% "Don't know" Adults: 21% "No" and 17% "Don't know".

Chilton indicates that a similar pattern of consumer misperception exists concerning the relationship between smoking and stillbirth. Among all teens, 44% did not know that smoking during pregnancy increases the risk of stillbirth; among adults, 48% did not know about the link.[81] As was the case for the relationship between smoking and miscarriage, smokers revealed a greater gap in knowledge than nonsmokers of the serious risk of stillbirth associated with maternal smoking during pregnancy. Forty-nine percent of teenage smokers doubted that maternal smoking increased the risk of stillbirth, while 57% of adults did not believe or know about the risk.[82]

In the Shor study at the University of New Hampshire,[83] 307 college students were asked a series of questions concerning the effects of smoking on pregnancy and infant health. Respondents were requested to rate statements according to the following scale: strongly believe, believe, neutral or uncertain, disbelieve, and strongly disbelieve.[84] When presented with facts concerning the health hazards to the fetus from maternal smoking during pregnancy, large proportions of this college-educated sample evidenced uncertainty or disbelief. Women who smoke during pregnancy have a significantly increased risk of spontaneous abortion or miscarriage.[85] When presented with this statement, 25% of the college students said they disbelieved it,[86] while another 48% said they were neutral or uncertain. In other words, almost three-fourths of the sample - 73% - did not know this fact.

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[80] Teens: 32% "No", 8% "Don't know", 12%, maternal smoking does not have any effect. Adults: 27% "No", 18% "Don't know", 13% maternal smoking does not have any effect.

[81] Teens: 24% "No", 10% "Don't know", 10% maternal smoking does not have any effect; Adults: 23% "No", 16% "Don't know, 10%, maternal smoking does not have any effect. Chilton, q. 39, 40(d).

[82] Teens: 27% "No", 10% "Don't know", 12% maternal smoking does not have any effect. Adults: 26% "No", 18% "Don't know", 13% maternal smoking does not have any effect. Chilton, q. 39, 40d.

[83] Shor, supra at 269.

Medical research demonstrates that babies of mothers who smoke are more likely to die within the first month after birth than babies of mothers who do not smoke.[87] About two-thirds of the college students questioned and three-quarters of the smokers did not believe or were uncertain about this fact.[88] Among smokers, 77% did not know about this increased risk of neonatal death for infants of smokers.[89]

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[84] A modified version of this methodology was employed as part of the 1980 Roper study.

[85] 1979 Surgeon General's Report, Ch. 8 at 32-35; 1980 Surgeon General's Report on Smoking and Women at 253.

[86] Twenty-three percent "Disbelieve", 2% "Strongly disbelieve". Shor, q. 25. The statement presented respondents was "Pregnant women who smoke have increased incidence of spontaneous abortion in comparison to nonsmokers."

[87] 1979 Surgeon General's Report, Ch. 8 at 41; 1980 Surgeon General's Report on Smoking and Women at 253.

[88] "Babies of smoking mothers have increased neonatal mortality (death within the first 28 days) in comparison to babies of non-smoking mothers." Shor, q. 24. Twenty percent of the sample disbelieved the statement, and 47% were neutral or uncertain, yielding a total of 67% doubting the fact.

The Shor study also suggests that the majority of college students may be unaware that women who smoke during pregnancy on the average have smaller babies. When presented with this fact, 62% of the sample, and 67% of the smokers, indicated doubt or disbelief.[90]

The Shor study relied solely on a sample of college students, and its results are therefore not generalizable to the larger population of all consumers.[91] Thus, it is possible that the results in the Shor study would very somewhat if a more representative sample were used.[92] In any event, the Shor study is supplemented by and consistent with the nationally projectable data available. The combined results of the 1980 Roper study, the Chilton study, and the Shor study represent one of the important findings of this report -- that a very substantial number of consumers, both men and women, do not know of the danger to the fetus and infant from maternal smoking.

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[89] Twenty-five percent "Disbelieve", 52% "Neutral or uncertain".

[90] Shor, q. 9. Overall: 21% "Disbelieve", 4% "Strongly disbelieve", 37% "Neutral or uncertain"; Smokers, 21% "Disbelieve", 7% "Strongly disbelieve" 39% "Neutral or uncertain." Evidence indicates that babies born to women who smoke during pregnancy are, on the average, 200 grams lighter than babies born to comparable women who do not smoke. 1980 Surgeon General's Report on Smoking and Women at 224.

[91] The authors acknowledge this in Shor, *supra* at 274.

[92] It is unclear what effect this sampling bias had, if any, on the results in Shor. On the one hand, it could be assumed that college students have greater knowledge than the general public in most areas and the bias here, if any, would result in an overestimation of the knowledge among the general public. The Commission has in the past assumed that college students are likely to have more knowledge than members of the general public. *Elliot Knitwear*, 59 F.T.C. 893 (1961). On the other hand, it could be argued that college-aged women, most of whom have never gone through a pregnancy themselves, are likely to have less knowledge of the danger of maternal smoking during pregnancy than older women. It is worth noting, however, that in the 1980 Roper study, women in the 18-29 year age group,

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92 (Footnote Continued)

presumably including most college students, evidenced more knowledge on these issues than women in older age groups.

VII. OTHER SMOKING-RELATED ISSUES

In each of the major medical areas discussed above, the data indicate significant gaps in consumer knowledge of the health hazards of smoking. In addition to these major medical areas, staff has analyzed data concerning consumer knowledge of several other areas. These areas are: the addictive nature of cigarette smoking; the presence of carbon monoxide in cigarette smoke; the relative safety of low "tar" and nicotine cigarettes; and the relationship between birth control pills, smoking, and heart attack.

A. Addiction

Many experts classify cigarettes as an addictive substance, as they do substances like narcotics, stimulants, sedatives or alcohol. This fact may be particularly important to non-smokers, especially teenage nonsmokers,[93] who may be considering whether to "experiment" with cigarettes. Evidence from Roper indicates that the *majority* of people do not think smoking is addictive. Half of the population felt smoking was merely a habit, and not an addiction;[94]

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[93] Evidence indicates that many teens believe cigarette smoking is "okay if they quit before it becomes a habit," and based on that belief, experiment with smoking. *Teenage Smoking: Immediate and Long-Term Patterns*, *supra* at 23, found that three out of five agreed with the above statement. Data show, however, that the vast majority of smokers who smoke as an experiment become regular users. If teens truly understood the addictive nature of cigarettes, they might be much more reluctant to 'experiment' with smoking.

[94] 1978 Roper, q. 32.

nearly half (49%) of all nonsmokers expressed doubt that smoking was addictive.[95]

B. Carbon Monoxide

Many researchers now believe that carbon monoxide may be one of the most harmful ingredients of the more than 2,000 known ingredients in cigarette smoke. CO is the substance in cigarette smoke which many medical experts believe to be most dangerous to fetal and infant health. In addition, many experts believe CO is the agent most likely to be responsible for the increased risk of heart disease among smokers.[96] Yet, the Chilton study reveals that many people are unaware that cigarette smoke contains carbon monoxide. When asked, "Does cigarette smoke contain carbon monoxide?", 51% of teens and 45% of adults either thought it did not or did not know.[97] Similar results were obtained by Roper in the 1980 study, where 53% of the sample and 56% of smokers did not know that "Cigarette smoke contains carbon monoxide, which is a dangerous gas."[98]

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[95] Forty-five percent said it was merely a habit; 1% felt it was neither a habit nor an addiction; and 3% answered "Don't know." 1978 Roper, q. 32. [Confidential Information Omitted]

[96] See, Ch. I, Sec: II, supra.

[97] Chilton, q. 43. Teens: 29% "No," 21% "Don't know"; Adults: 19% "No", 26% "Don't know."

[98] 1980 Roper, q. 30. Total: 31% "Don't know if it's true," 13% "Think it's not true", 10% "Know it's not true". Smokers: 24% "Don't know if it's true", 19% "Think it's not true", 13% "Know it's not true."

C. Low "Tar" And Nicotine Cigarettes

"Tar" and nicotine are among the most harmful ingredients in cigarettes. However, they are not the only harmful ingredients. The 1979 and 1981 Surgeon General's Reports both conclude that there is insufficient evidence to conclude that smoking low "tar" and nicotine cigarettes is safe, or even significantly safer than regular cigarettes.[99]

In the 1980 Roper study, respondents were presented with the following false statement: "It has been proven that smoking low-tar, low-nicotine cigarettes does not significantly increase a person's risk of disease over that of a nonsmoker." More than one out of three -- 36% -- of smokers said they knew or thought this was true, while another 32% of the smokers said they didn't know if it was true or not.[100] This evidence suggests that many smokers falsely believe that smoking is not dangerous to them if they smoke low-"tar" and low-nicotine cigarettes.[101]

D. Birth Control Pills

Smoking is especially dangerous for women who also take birth control pills. Smoking, alone, doubles the risk of heart attack over that of non-smokers. Studies have also found that the risk of heart attack among women during child bearing age is approximately doubled by the use of estrogen containing oral contraceptives. However, women who smoke and take the pill have approximately ten times the risk of heart attack than that of women who do neither.[102] In Chilton, respondents were asked whether the following statement was true: "If a woman smokes and uses birth control pills, it doesn't increase her chances of getting a heart attack." Twenty-six percent of teens and 30% of adults either believed this false statement, or answered "don't know".[103] Smokers evidenced a greater lack of knowledge in this area than non-smokers. Thirty percent of teenage smokers and 36% of adult smokers were unable to answer the question correctly.[104]

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[99] See, Ch. I, Sec. II, supra; 1981 Surgeon General's Report.

[100] 1980 Roper, q. 30. 9% "Know it's true", 27% "Think it's true."

[101] [Confidential Information Omitted]

Consumer knowledge in this area was further tested in the 1980 Roper study. Respondents were presented with the statement: "A woman who takes birth control pills further increases her risk of getting a heart attack if she also smokes." Thirty-six percent of the women polled did not know this fact.[105] These data constitute another important finding of Chilton and Roper: consumer knowledge of the relationship between smoking, birth control pills, and heart attack is low.

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[102] 1980 Surgeon General's Report on Smoking and Women at 114-116; 1979 Surgeon General's report, Ch. 4 at 60.

[103] Teens: 17% "True", 9% "Don't know"; Adults: 14% "True", 16% "Don't know." Chilton, q. 53.

[104] Teens: 18% "True," 12% "Don't know", Adults: 17% "True", 19% "Don't know."

[105] 1980 Roper, q. 30. 28% "Don't know if it's true", 6% "Think it's not true," 2% "Know it's not true." Respondents who did know of the risk were then asked to quantify the risk. Respondents were asked how much more likely a woman who smokes and takes birth control pills is to have a heart attack than one who does neither. One sample was given the choices: (1) less than twice as likely (2) twice as likely (3) five times as likely (4) ten times as likely (the (Continued)

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105 (Footnote Continued)

correct answer), and (5) twenty times as likely. The vast majority of respondents underestimated the risk, and the response most often given was "twice as likely". To test for bias from choice order and from the choices presented, a separate sample was given these choices: (1) five times (2) ten times (3) twenty times (4) thirty times (5) fifty times. Predictably, more people chose the correct answer in this sample than in the other one. However, even given these choices the response most often given was the lowest - "five times" - and more than twice as many respondents underestimated the risk than overestimated. 1980 Roper, q. 32.

VIII CONCLUSION

The data indicate serious, significant gaps in consumer knowledge of the hazards of cigarette smoking. While most people know that smoking is somehow hazardous to health, substantial numbers of people evidence a considerable gap in knowledge concerning the overall impact of smoking on the risk of dying and illness. For the substantial number of people who have only a vague general awareness of the hazards of smoking without knowing the specific hazards and risks, the following 1964 conclusion of the Commission is worth reconsidering:

> ...[T]he argument that everyone is aware of the health hazards of smoking fails to take adequate account of the existence of different levels of awareness. To be remotely or dimly aware of a subject is not the equivalent of having the kind of knowlege upon which people normally act. Much of the publicity concerning the health implications of cigarette smoking is mere hearsay. Many people are aware that it has been said that smoking is harmful; but this is not the same as _knowing_ that smoking is harmful.
> 1964 Cigarette Rule, _supra_ at 8360 (emphasis added).

In fact, the data discussed in this chapter suggest that many consumers do not know enough about the health effects of smoking to know _how dangerous smoking is_.

The results, discussed in detail above, indicate that many are unaware of the _existence of the relationship_ between smoking and some of its most serious and widespread health consequencs, such as heart disease -- the number one cause of smoking-related deaths -- and miscarriage and stillbirth. As the data indicate, over 30% of the public is unaware of the relationship between smoking and heart disease. Nearly 50% of all women do not know that smoking during pregnancy increases the risk of stillbirth and miscarriage. Approximately 30% of those polled do not know about the relationship between smoking, birth control pills and the risk of heart attack. Some of the health consequences of smoking, such as lung cancer and emphysema, are more well known. However, even for lung cancer, the most well known health effect, some substantial gaps in consumer knowledge are evident.

The data also indicate that substantial numbers seriously misunderstand and underestimate the increased risk of suffering these health problems as the result of smoking. For example, although 80 to 85% of all lung cancer cases are smoking-related and a smoker is about ten times as likely to develop lung cancer as a non-smoker is, the survey data discussed in this chapter indicate that over 40% of those polled did not know that smoking caused most cases of lung cancer and nearly one-quarter of those polled did not even know it causes many cases. Similarly, although the evidence indicates that smoking causes about 70% of all cases of chronic obstructive lung disease (emphysema and chronic bronchitis), approximately 60% of the public does not know that most cases of emphysema are smoking-related, and one-third of those polled are even unaware that many cases of emphysema are smoking-related. In addition, over 50% of the population does not know that smoking causes many as opposed to just a few cases of heart attack.

As the evidence in the section on "Overall Risk" demonstrates, many more consumers seriously underestimate the severity or increased risk of dying from these smoking-related illnesses. Among those polled in the 1980 Roper study, 30% of the population and 41% of the smokers did not know that a typical thirty-year old male shortened his life expectancy at all by smoking. Approximately one-half of those polled greatly underestimated the risk of death from lung cancer by selecting responses indicating that they believed that substantially fewer than one-half of those who suffer from lung cancer die from the disease.

Finally, the survey data indicate that a large number of people do not believe that the health consequences of smoking have personal relevance to themselves. Thus, although the medical evidence clearly indicates that smoking only a few cigarettes a day can be harmful, nearly one-third of those polled and 40% of smokers polled believed that only heavy smoking is dangerous. Similarly, although the Surgeon General has concluded that low "tar" cigarettes have not been shown to be safe, over one-third of the smokers polled falsely believe that it has been proven that by smoking low "tar" cigarettes, smokers do not significantly increase their health risks over nonsmokers. Thus, many smokers who do not consider themselves to be "heavy" smokers and many who believe that low "tar" cigarettes are safe incorrectly believe that the health risks associated with smoking do not apply to them. Another example may be particularly important for nonsmokers, especially young people deciding whether to experiment with smoking. Although many experts now classify cigarettes as an addictive substance, many teenagers believe smoking is okay if they quit before it becomes a habit and approximately 50% of all those polled did not know that smoking is addictive for many people.

In conclusion, the data discussed above indicate that a substantial portion of consumers do not understand the health hazards of smoking.

CHAPTER IV. ROLE OF THE FEDERAL TRADE COMMISSION*

I. CIGARETTE ADVERTISING HAS LONG BEEN A CONCERN OF THE FEDERAL TRADE COMMISSION

Cigarette advertising has been a major concern of the Federal Trade Commission since it began examining cigarette advertising in the late 1930's. The Commission's first actions sought to prevent cigarette companies from making unsupported claims about the medical and other benefits of particular brands.[1] Between 1945 and 1960, the Commission issued seven cease and desist orders prohibiting various false claims in cigarette advertising.[2] In 1955, however, the Commission shifted its attention to industry-wide regulation and promulgated Cigarette Advertising Guides.[3] In 1960, the Commission obtained agreements from the leading cigarette manufacturers to eliminate from their advertising unsubstantiated representations of "tar" and nicotine content.[4]

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* In addition to the information in this Chapter, the Commission relied upon information in a confidential report evaluating the readership of cigarette advertisements, a confidential copy test, and two surveys. This information, which was provided by the cigarette companies pursuant to subpoena has been deleted from the version of the report released to the public.

[1] *E.g.*, *Julep Tobacco Co.*, 27 F.T.C. 1637 (1938); *Green River Tobacco Co.*, 27 F.T.C. 1547 (1938).

[2] *R. L. Swain Tobacco Co.*, 41 F.T.C. 312 (1945); *P. Lorillard Co.*, 46 F.T.C. 735, *order modified*, 46 F.T.C. 853, *aff'd*, 186 F.2d 52 (4th Cir. 1950); *R. J. Reynolds Tobacco Co.*, 46 F.T.C. 706 (1950), *modified*, 192 F.2d 535 (7th Cir. 1951), *on remand*, 48 F.T.C. 682 (1952); *American Tobacco Co.*, 47 F.T.C. 1393 (1951); *Philip Morris & Co., Ltd.*, 49 F.T.C. 703 (1952), *vacated and remanded on motion of Commission, complaint dismissed on affidavit of abandonment*, 51 F.T.C. 857 (1955); *Liggett & Myers Tobacco Co., preliminary injunction denied*, 108 F. Supp. 573 (S.D.N.Y. 1952), *aff'd mem.*, 203 F.2d 955 (2d Cir. 1953), *decision of Commission*, 55 F.T.C. 354 (1958); *Brown & Williamson Tobacco Corp.*, 56 F.T.C. 956 (1960) (consent order).

The Commission responded swiftly to the 1964 Surgeon General's Report,[5] taking its first formal action that same year to require that cigarette manufacturers warn consumers of the health hazards of smoking. Given the "mounting evidence...of the very grave hazards to life and health involved in cigarette smoking," the Commission found that failure of the manufacturers to warn consumers of the danger constituted an unfair and deceptive practice in violation of Section 5 of the Federal Trade Commission Act.[6] The Trade Regulation Rule promulgated by the Commission would have required all cigarette packages and advertisements to disclose clearly and prominently that

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[3] These Guides prohibited, in cigarette advertising and labeling, representations as to the presence or absence of any physical effects from cigarette smoking, unsubstantiated claims about nicotine, "tar" or other components of cigarette smoke, and misleading implications concerning the health consequences of smoking.

[4] 2 Trade Reg. Rep. (CCH) ¶ 7853.51.

[5] Office on Smoking and Health, U.S. Department of Health, Education and Welfare, Smoking and Health: A Report of the Surgeon General (1964) ("1964 Surgeon General's Report").

[6] The 1964 Cigarette Rule at 8324, 8325, 8356.

"cigarette smoking is dangerous to health and may cause death from cancer and other diseases."[7]

In 1965, however, Congress pre-empted the F.T.C. action by enacting legislation requiring all cigarette packages distributed in the United States to include the statement: "Caution: Cigarette Smoking May Be Hazardous To Your Health."[8] In addition, the Cigarette Act prohibited the F.T.C. from requiring any other statement on the cigarette package, and precluded, until July 1, 1969, any government requirement of a disclosure regarding smoking and health in cigarette advertisements. The Act also directed the Commission to submit an annual report to Congress on cigarette advertising.

Shortly thereafter, the Commission succeeded in finding an acceptable uniform testing system for measuring the "tar" and nicotine content of cigarettes. In 1966, a letter was sent to U.S. cigarette manufacturers approving their factual statements of "tar" and nicotine content, if such statements were supported by tests conducted in accordance with the approved method.[9] In 1967, the Commission activated its own laboratory to analyze the "tar" and nicotine content of cigarette smoke, and, pursuant to a request from the Chairman of the Senate Commerce Committee, then F.T.C. Chairman

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[7] Id., at 8325.

[8] Federal Cigarette Labeling and Advertising Act ("Cigarette Act"), 15 U.S.C. §§ 1331 et. seq. (1965).

[9] 4 Trade Reg. Rep. (CCH) ¶ 39,012.70. Letter from Federal Trade Commission to major cigarette manufacturers and to Robert B. Meyner, Administrator of the Cigarette Advertising Code (March 25, 1966).

Paul Rand Dixon agreed to test and report periodically to Congress the "tar" and nicotine content of various cigarettes. Test results have been published on a regular basis since that time.

As the congressionally mandated moratorium approached termination in mid-1969, the Commission proposed a modified version of the 1964 Rule that would require all cigarette advertisements to carry the following message: "Warning: Cigarette Smoking Is Dangerous To Health and May Cause Death From Cancer, Coronary Heart Disease, Chronic Bronchitis, Pulmonary Emphysema, and Other Diseases".[10] Congress, however, then amended the message on cigarette packages to read: "Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous To Your Health."[11] Congress also banned cigarette advertisements from the broadcast media after January 1, 1971, and prohibited the Commission from taking any action on its proposed rule prior to July 1, 1971.[12]

In the interim, the Commission proposed a Trade Regulation Rule that would have made it an unfair or deceptive practice for cigarette companies to fail to disclose in their advertising the "tar" and nicotine content of the advertised brand, based on the most recent

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[10] 34 Fed. Reg. 7917 (1969).

[11] 15 U.S.C §§ 1331 et. seq. (1970).

[12] Id. As a result of the Congressional action prohibiting cigarette advertising from the broadcast media, the Commission decided not to pursue its proposed rule.

FTC test results.[13] Subsequently, the Commission indefinitely suspended the proceeding to allow the manufacturers to implement a voluntary plan to disclose "tar" and nicotine levels in their advertising. Since 1971, all manufacturers have made disclosures in accordance with this plan.

Later that year, the Commission announced its intention to file complaints against the cigarette companies, alleging that advertising without a clear and conspicuous disclosure that smoking is dangerous to health would be "false and misleading" and constitute an "unfair practice." Lengthy negotiations between the Commission and the six major cigarette manufacturers culminated in 1972 in consent orders requiring all cigarette advertising to display clearly and conspicuously the same warning that Congress already had required on cigarette packages.[14]

Four years later, in May 1976, the Commission again renewed its investigation of cigarette advertising to determine whether its prior actions, or the actions of the cigarette industry in the interim, had _effectively_ remedied the deceptive practices which previously had caused the Commission to conclude that remedial action was necessary. This latest investigation was begun by issuance of subpoenas to industry members and their advertising agencies. In January 1979, following a two and one-half year legal proceeding, the Commission won an enforcement action compelling compliance with the subpoenas.[15]

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[13] 35 Fed. Reg. 12671 (1970).

[14] _Lorillard_, _et al_, 80 F.T.C. 455 (1972). In 1975, the Commission voted to seek civil penalties against the six major cigarette manufacturers for violation of the orders. This litigation is still pending in the U.S. District Court for the Southern District of New York. _FTC v. American Brands et al_. C.2180-85 (S.D.N.Y. 1980).

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[15] F.T.C. v. Carter, 464 F. Supp. 633 (D.D.C. 1979).

II. STAFF BELIEVES THE CURRENT WARNING IS INEFFECTIVE

The current health warning was included in cigarette advertisements as a result of consent orders signed by the six major cigarette companies and the Commission in 1972.[16] The current investigation was begun to determine whether the deceptive practices which previously had caused Congress and the Commission to act have been effectively remedied. Therefore, the effectiveness of the current warning as a remedy had to be reexamined. After careful examination, the staff believes that the current warning is not effective in curing the deception in cigarette advertising, nor does it provide sufficient information to permit consumers to assess the health risks of smoking accurately.[17]

There are two major objective indicia of the ineffectiveness of the current warning, discussed in detail below. First, as the staff's investigation of consumer knowledge has demonstrated, a substantial portion of the public remains uninformed about the hazards of smoking. Second, tests conducted by the FTC [Confidential Information Omitted] indicate that the warning is neither noticed nor read by the vast majority of people. The underlying causes of the current warning's ineffectiveness are discussed below.[18]

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[16] Lorillard, et al., 80 F.T.C. 455 (1972).

[17] See Ch. III, supra.

[18] The reasons discussed in this section do not constitute the only reasons for the current warning's ineffectiveness. As will be explained in detail, infra at Ch. V, the unchanging size and shape of the current warning contribute to its ineffectiveness.

The most persuasive evidence that the current warning is not effective is the evidence indicating that a substantial portion of the population is uninformed about the health hazards of smoking. Significantly, as the evidence indicates, for millions of consumers, the current warning has not provided them with or stimulated them to learn the more specific health information needed to understand the magnitude and the nature of the health hazards of smoking.[19] Also, despite the explicit language of the Surgeon General's warning, 17% of all smokers and 24% of heavy smokers still do not know the very basic fact that smoking is hazardous to health.[20]

Evidence demonstrating that the warning is simply not noticed or read by the vast majority of consumers is another indicia of the ineffectiveness of the current warning. In 1978, the Starch Message Report Service tested the readership of cigarette ads in 24 different magazines for the FTC. Only 2.4% of adults exposed to the tested cigarette ads read the Surgeon General's warning. [Confidential Information Omitted]

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[19] See, Ch. III, supra.

[20] Gallup Opinion Index at 20; see, Ch. III, Sec II, supra. See also pp. 3-3 and 3-4.

[21] [Confidential Information Omitted]

[22] [Confidential Information Omitted]

Consumers agree that the current warning is inadequate. Almost two of every three questioned during the 1978 Roper survey for the Tobacco Institute felt that the current warning is not "adequate" and indicated that they preferred a health warning that describes the specific health risks of smoking.[23]

It is important to understand why the current warning is no longer noticed. The reasons underlying the warning's ineffectiveness, however, go beyond noticeability. A warning may be noticed much more often than the current warning and still be ineffective. Even the ability of consumers to recall the content of the message correctly is not necessarily an accurate gauge of its effectiveness. An effective warning is one that permits consumers to recall and consider its message spontaneously and consciously without prompting at the time a purchasing decision is made.[24] Studies have shown that people do not typically spontaneously recall or consider all of the relevant information stored in their memory in making judgments or decisions. When deciding to smoke, for example, consumers do not ordinarily run through a check list of all they remember or know about the positive and negative aspects of smoking. Rather, only a small subset of the information is actually spontaneously recalled or considered in making their decision, i.e., the information that happens to be most accessible at the time.[25] For four basic reasons, described in detail below, the current warning is not accessible, and, therefore, cannot be effective. They are: (1) the current warning is overexposed and worn out; (2) the current warning is no longer novel and presents no new information; (3) the abstract nature of the current warning makes it more difficult to remember; (4) the current warning is not likely to be perceived as personally relevant.

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[23] 1978 Roper Report, q. 25.

[24] Cohen & Srull, Information Processing Issues Involved in the Communication and Retrieval of Cigarette Warning Information, Report Prepared for the Federal Trade Commission, November 1980 ("Cohen and Srull") at 11.

In contrast to the current warning, cigarette advertisements present positive information about smoking in a highly effective manner. They communicate the desirable aspects of smoking in a variety of attention-getting formats. Having attracted the reader's attention, the ads present positive information about smoking through numerous concrete image-provoking, personalizable themes, each theme presented in many different ways. Information that is distinctive in form, novel or varying in content, concrete, and personalizable is precisely the type of information that people are most likely to notice, think about and spontaneously recall.

Wearout. The Surgeon General's warning fails in part because it has been overexposed; it has been presented in a small inconspicuous rectangle and with unchanged wording in every cigarette advertisement for almost a decade. Although repetition of a message initially contributes to improved long-term recall of that message,[26] unless the message is varied it will soon become so familiar that it will "wear out" and no longer be consciously perceived.[27] Whether or not the current warning was ever widely noticed, the evidence indicates that it is no longer read or noticed by most Americans exposed to cigarette advertising, which is not surprising given that neither its shape, nor its wording ever change.[28]

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[25] See, e.g., Tversky & Kahneman, Availability: A Heuristic For Judging Frequency and Probability, 5 Cognitive Psychology 207-32 (1973); Tversky & Kahneman, Judgment Under Uncertainty: Heuristics and Biases, 185 Science 1124-31. (1974)

Novelty. One of the most potent factors in increasing the likelihood that a piece of information will be spontaneously retrieved is novelty.[29] Studies have shown that information that is novel or unexpected is likely to capture one's attention, is processed more extensively, and subsequently is much more likely to be recalled than information that is redundant or expected to appear in a given context. Hundreds of studies have demonstrated that any technique that increases an item's novelty enhances subsequent recall of that item.[30]

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[26] *See, e.g.*, Sawyer & Ward, "Carryover Effects in Advertising Communication: Evidence and Hypotheses from Behavioral Science," Clarke, ed. *Cumulative Advertising Effects: Sources and Implications*, 1977.

[27] Kaufman, *Memory Without Recall, Exposure Without Perception*, J. of Advertising Research (Aug. 1977). *See, e.g.*, Craig et al., *Advertising Wearout: An Experimental Analysis*, J. of Marketing Research (Nov. 1976); Greenberg et al., *Television Commercial Wearout*, J. of Advertising Research (Oct. 1973); Appel, *Advertising Wearout*, J. of Advertising Research (Feb. 1971).

[28] Zuckerman, *Use of Consensus Information in Prediction of Behavior*, Journal of Experimental Social Psychology, 163-171 (1978); Nisbett, Borgia, Crandall & Reed, *Popular Induction: Information is Not Necessarily Informative*, in Cognition and Social Behavior Carroll & Payne (eds.) 1976. Wyer & Hartwick, *The Role of Information Retrieval and Conditional Inference Processes in Belief Formation and Change*, in Advances in Experimental Social Psychology J. Berkowitz (ed.) (in press).

[29] Cohen & Srull, *supra*, at 12.

The least novel, most redundant element in any cigarette advertisement is the Surgeon General's warning. Neither its content nor its form ever varies. Novel information not only captures more attention and is better recalled than redundant information, but it does so at the expense of the redundant information.[31] For example, researchers have found that novel items not only showed enhanced recall but the immediately surrounding items showed unusually *low* levels of recall.[32] These results suggest that one's attention is drawn to novel information. However, since one's attention and processing capacity is limited, this necessarily means that *less* attention is paid to immediately surrounding information. Since advertisements are continually changing and often contain novel verbal and pictorial material, it is not surprising to find that many people report not even seeing the warning label when looking at standard advertisements. Thus, unless the current warning is changed somehow to make it more novel, it will become increasingly less effective.

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[30] This is the well known "von Restorff effect" based on the memory research by von Restorff in the 1930's. Hundreds of studies have since replicated this effect, *see, e.g*, Hastie, *Schematic Principles in Human Memory*, in Higgins, Herman & Zanna (eds.), Social Cognition: The Ontario Symposium on Personality and Social Psychology (Erlbaum, in press); Wallace, *Review of the Historical, Empirical, and Theoretical Status of the Von Restorff Phenomenon*, 63 Psychological Bulletin, 410-24 (1965).

[31] *See, e.g.*, Newman & Saltz, *Isolation Effects: Stimulus and Response Generalization as Explanatory Concepts*, 55 Journal of Experimental Psychology, 467-72 (1958).

[32] *Id*.

Concrete and abstract information. The abstract and general nature of the current warning is another factor contributing to its ineffectiveness. Consumers are unlikely to consider abstract general information like the information contained in the current health warning in their smoking decisions.[33] Concrete information generally refers to objects or events that are readily transformed into mental images. In contrast, abstract information generally refers to abstract concepts that are not readily tranformed into mental images. A picture is obviously concrete since it already contains a specific visual image; words may also be concrete. For example, the words "smoke," "heart" and "cigarette" are concrete, as it is easy to form mental images of such objects. On the other hand, words such as "hazardous" or "health" are very abstract and difficult to transform into mental images.

The ability of certain forms of communication to be transformed into mental images has a number of important consequences. First, concrete information is better remembered than abstract information. Pictures are better remembered than words, and concrete words are better remembered than abstract words.[34] Therefore, the current warning is much less likely to be remembered that the other messages communicated in cigarette advertising. Many cigarette advertisements contain rich photographic imagery. In addition, the copy is usually dramatic and concrete. In contrast, the present warning is entirely abstract. It contains words like "health," "hazardous," and "concluded" that are extremely difficult to visualize. It also fails to provide any concrete evidence on which the abstract conclusion is based. Since consumers are not likely to remember the abstract warning, it is not going to be accessible to them when they make a decision to buy or smoke cigarettes.

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[33] See, e.g., Nisbett, Borgida, Crandall & Reed, Popular Induction: Information is Not Necessarily Informative, In Carroll & Payne (eds.), Cognition and Social Behavior (Erlbaum 1976).

Second, numerous studies indicate that people rely on concrete information much more than on abstract information in making judgments and decisions.[35] Indeed, it has been shown that there is a dramatic overreliance on concrete information accompanied by a corresponding underreliance on abstract information.[36] For example, people ignore abstract descriptive information about a population of people to predict the behavior of an individual, but often use the concrete behavior of an individual to predict characteristics of the entire population.[37] Similarly, it has been shown that when people estimate the risk associated with various activities or events, abstract statistical summaries are largely ignored, while vivid individual cases are weighed quite heavily.[38]

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[34] *See, e.g.*, Mandler & Johnson, *Some of the Thousand Words a Picture is Worth*, 2 Journal of Experimental Psychology: Human Learning and Memory 529-40 (1976); Paivio, *Imagery and Verbal Processes* Holt, Rinehart & Winston (1976).

[35] *See, e.g.*, Nisbett & Ross, *Human Inferences: Strategies and Shortcomings of Social Judgment* (Prentice-Hall 1980); Nisbett, Borgida, Crandall & Reed, *Popular Induction: Information is Not Necessarily Informative* in Carroll & Payne (eds.) Cognition and Social Behavior (Erlbaum 1976).

[36] *See, e.g.*, Borgida & Nisbett, *The Differential Impact of Abstract vs. Concrete Information On Decisions*, 7 Journal of Applied Social Psychology 258-71 (1977).

Thus, the concrete information contained throughout most cigarette ads lessens even further the impact of the abstract information contained in the health warning. The ads are rich in thematic imagery associating smoking with warmth, friendliness, outdoor activities, athletics, and individualism. They are filled with vigorous, attractive, healthy-looking people living energetic lives full of social acceptance, success, and athletic achievement, free from any smoking hazards. Individuals seeing these cigarette ads are much more likely, therefore, to use the concrete positive images of smoking in deciding whether or not to smoke than they are the abstract general health warning.

Personal relevance of information. Information which consumers perceive as having personal implications and relevance to themselves is most likely to be noticed, remembered and considered. Abstract information is more likely to be perceived as personally irrelevant than concrete information. There is a large difference between being aware of the statement that "smoking is dangerous" and believing that "my smoking will injure me."[39] The former is seen as an abstract statement which is difficult to interpret in terms of one's personal experiences. In contrast, the evidence indicates that relating a piece of information to oneself induces the deepest levels of memory.[40] The current warning is not likely to be seen as personally relevant. It is extremely abstract. As such, it is less likely to be spontaneously recalled and less likely to be considered in any smoking decision.[41]

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[37] *Id.*; *see also*, Hamill, Wilson & Nisbett, *Insensitivity to Sample Bias: Generalizing from Atypical Cases*, 39 Journal of Personality and Social Psychology, 578-89 (1980).

[38] Slovic, Fischoff & Lichtenstein, *Cognitive Processes and Societal Risk Taking* in Carroll & Payne (eds.) Cognition and Social Behavior (Erlbaum 1976).

In conclusion, all the available evidence indicates that the current warning is an ineffective remedy to the possible deception by omission in cigarette advertising. Staff has considered the reasons for this ineffectiveness in its discussion of alternative methods for more effectively providing consumers with additional health information about the health risks of smoking, *See*, Ch. V., *infra*.

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[39] *See, e.g.*, Fishbein, Consumer Beliefs and Behavior With Respect to Cigarette Smoking: A Critical Analysis of the Public Literature, F.T.C. Annual Report to Congress on Cigarette Advertising for the Year 1977, Appendix A.

[40] Rodgers, Kuiper & Kirker, *Self-Reference and the Encoding of Personal Information*, 35 Journal of Personality and Social Psychology, 677-88 (1977).

[41] There is also a great deal of evidence that people are more likely to dismiss as irrelevant evidence that is highly discrepant with their own behavior or beliefs. In creating a remedy to any possible deception, consideration must be given to this problem.

III. LEGAL DISCUSSION: CURRENT CIGARETTE ADVERTISING MAY VIOLATE THE SECTION 5 PROHIBITION AGAINST DECEPTIVE ADVERTISING[42]

A. Introduction

If the Commission determines that it is necessary and appropriate for it to take some action to remedy the problem discussed in this report, it will be necessary to determine whether the problem discussed violates the Federal Trade Commission Act. Section 5 of the Federal Trade Commission Act has long been interpreted to proscribe all forms of deceptive advertising. The test for unlawful deception under Section 5 is whether a "substantial segment of the purchasing public is likely to be deceived."[43] It is not necessary to prove that an advertisement has actually misled the public to justify a finding that the ad is unlawfully deceptive. It is sufficient for the Commission to find that the advertisement has the tendency or capacity to deceive.[44] The determination of the meaning of an ad and its tendency to mislead consumers also need not depend on exhibits, testimony, or a survey of consumers' perceptions, although the Commission considers such evidence, whenever available, so that its judgment will be as informed as possible.[45] Based on its own expertise, the Commission can determine whether an advertisement is deceptive from a review of the advertisement itself.[46] In so doing, the Commission may consider the net impression the ad may convey to the general populace.[47] The meaning of an advertisement and its tendency or capacity to deceive are questions of fact to be determined by the Commission.[48]

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[42] Section 11 of the Federal Trade Commission Improvements Act of 1980, Public Law 96-252, prohibits the Commission from initiating any new rulemaking proceeding which regulates commercial advertising on the basis that such commercial advertising conutitutes an "unfair" act or practice. In light of the fact that one of the possible courses of action the Commission may at some time take as a result of the information contained in this report is to initiate a rulemaking proceeding, the staff report does not evaluate whether current cigarette advertising practices may be "unfair".

[43] 1964 Cigarette Rule, *supra* at 8350.

[44] *Beneficial Corp. v. F.T.C.*, 542 F.2d 611, 617 (3rd Cir. 1976); *Charles of the Ritz Dist. Corp. v. F.T.C.*, 143 F.2d 676, 680 (2d Cir. 1944).

Even where no false statement is made, deception may occur by the advertiser's failure to disclose material facts in a situation in which there is a disparity between the actual consequences which may result from the use of the advertised product and the expectations of a substantial segment of the public about the consequences of using the product.[49] An advertiser's failure to disclose material facts in these circumstances is fully equivalent to deception by misleading statements or suggestions.[50]

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[45] *The J.B. Williams Co. v. F.T.C.*, 381 F.2d 884, 889 (6th Cir. 1967); *Carter Products Inc. v. F.T.C.*, 323 F.2d 523, 528 (5th Cir. 1963).

[46] *Carter Products, Inc. v. F.T.C.*, 323 F.2d at 528.

[47] *Id.*

[48] *Kalwajtys v. F.T.C.*, 237 F.2d 654, 656 (7th Cir. 1956), *cert. denied*, 352 U.S. 1025 (1957); *Gulf Oil Corp. v F.T.C.*, 150 F.2d 106, 108 (5th Cir. 1945).

[49] 1964 Cigarette Rule, *supra*, at 8352.

However, the Commission's concern about the inclusion of information in advertisements is limited to situations in which the advertiser's failure to disclose certain information constitutes "an unfair or deceptive act or practice" within the meaning of Section 5 of the Federal Trade Commission Act. Advertisements need not contain every piece of information about the product being advertised. In fact, the situations in which the Commission has required informational disclosures in advertisements and the type of information which has been required to be disclosed have been carefully limited. Thus, it is necessary to analyze each of the elements of the law of deception by omission carefully to determine what information the law requires to be included.

B. Elements of the Law of Deception by Omission as Applied to Cigarette Advertising.

In the Statement of Basis and Purpose for the 1964 Cigarette Rule, the Commission articulated three elements of deception by omission:

> (1) Certain facts about a product or the consequences of its use must not be adequately disclosed in the advertisements.
>
> (2) The information which is not disclosed must be material. A material fact has traditionally been defined as one "likely to affect the average consumer in deciding whether to purchase the advertised product."

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[50] 1964 Cigarette Rule, *supra*, at 8351; *P. Lorillard Co. v. F.T.C.*, 186 F.2d 52, 58 (4th Cir. 1950); *Simeon Management Corp. v. F.T.C.*, 579 F.2d 1137, 1145 (9th Cir. 1978).

(3) A substantial portion of consumers must be aware of or hold false expectations about the nondisclosed material facts.[51]

In addition, as we discuss in detail at the conclusion of this section, the Commission has been particularly concerned when one or more of the following factors is present: (a) the product is hazardous to health, (b) particularly vulnerable groups of consumers are affected, and (c) the deception is aggravated by continuous, massive advertising.

Applying the elements of the law of deception by omission to current cigarette advertising leads the staff to conclude that it may be deceptive. Little discussion is needed to establish the existence of the first element.

It is clear that current cigarette advertising fails to disclose adequate facts about cigarettes and the health consequences of their use. The ads do not reveal any of the information about the specific hazards of smoking, the severity of these risks or the smokers' increased probability of suffering these harmful effects. As we previously noted, while the current warning may have represented a reasonable remedial effort a decade ago based on the evidence then available, new evidence appears to indicate that the current warning is ineffective. The remaining two elements of the law of deception by omission are discussed in further detail below.

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[51] 1964 Cigarette Rule, supra at 8351.

1. *A "substantial segment" of the population has a lack of knowledge of or holds false beliefs about the hazards of of cigarette smoking*.[52]

To find deception

> [t]he Commission is not required to establish that a preponderance of the public is deceived. It is only necessary to establish...[that the ads have the capacity to deceive] *any group of buyers* even though they may be more susceptible to misrepresentations of the seller (intended or unintended) than a majority of buyers.[53]

In cases such as this involving health and safety, the Commission has found that the "substantial portion" requirement has been met when as few as 9% of the public was misled. However, the data in this report indicate that a much larger portion of the public lacks sufficient knowledge of the hazards of cigarette smoking. Therefore, as part of this investigation, the Commission need not decide whether, today, there would be a violation of Section 5 if as few as 9% of the public was misled. In addition, although the cases cited in the following analysis involve advertisements containing false statements, the same rationale which led the Commission to reach its conclusion in these cases applies to cases of deception by omission. Therefore, it is appropriate for the Commission to apply the same legal principles.[54]

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[52] 1964 Cigarette Rule, *supra*, at 8350; *Rhodes Pharmacal Co. v. F.T.C.*, 208 F.2d 382 (7th Cir. 1953); *Bristol-Myers Co., et al.*, 85 F.T.C. 688 (1975).

[53] *Benrus Watch Co. Inc., et al.*, 64 F.T.C. 1018, 1032 (1964).

[54] 1964 Cigarette Rule, *supra* at 8351; *Simeon Management Co. v. F.T.C.*, 579 F.2d at 1145.

In *Rhodes Pharmacal Co. v. F.T.C.*, 208 F.2d 382 (7th Cir. 1953), a Commission finding of deception in advertising of a claimed "cure" for arthritis was upheld, although evidence indicated that only 9% of the public interpreted the ad to suggest the product "cured" arthritis. Similarly, in a case involving safety claims for automobile tires, the U.S. Court of Appeals for the Sixth Circuit refused "to overturn the deception findings of the Commission if the ad...misled 15% (or 10%) of the buying public." *Firestone Tire and Rubber Co. v. FTC*, 481 F.2d 246, 249 (6th Cir. 1973).

In several other cases, the Commission has reached similar results. In *Benrus Watch Co. Inc., et al.*, 64 F.T.C. 1018 (1964), the company argued that the public was not misled by Benrus' policy of including an inflated "manufacturer's suggested price" on its watches, because 86% of the public, according to survey data, "knew" that retail prices commonly varied from manufacturers' suggested prices. The Hearing Examiner, whose findings were adopted by the Commission, concluded that:

> ...assuming that 86% of the public would not be deceived by the ticketed price because they knew prices were variable, the remaining 14% [who did not know] would apparently be deceived by the ticketed price. [The survey] therefore indicates unequivocally that a substantial segment of the public would be deceived...
> 64 F.T.C. at 1032.

In *ITT Continental Baking Co. Inc., et al.*, 83 F.T.C. 865 (1973), *modified*, 83 F.T.C. 1105 (1973), *aff'd* 532 F.2d 207 (2nd Cir. 1976), the Commission found that Wonder Bread ads with misleading claims concerning nutrition and growth had the capacity to deceive a "substantial portion" of the public based upon surveys indicating that 10% of the public in 1970 and 9% in 1971 were misled. In

Bristol-Myers Co. et al., 85 F.T.C. 688 (1975), the Commission, in dismissing a deception action on other grounds, agreed that survey results which indicated beliefs of "probably somewhere between 14% and 33%" of the population satisfied the "substantial portion of the public" standard. 85 F.T.C. at 744. In *Elliot Knitwear, Inc., et al.*, 59 F.T.C. 893 (1961), the Commission held that labeling practices which are likely to deceive 13% of the population are actionable under Section 5.

As defined by previous cases, the "substantial portion" standard is satisfied in the case of cigarette advertising. While staff has proceeded on the assumption that most Americans are generally aware that smoking may be hazardous to health, the data show that some still are not and that among smokers, many are still not aware of this fact. The data indicate that 17% of smokers don't believe this basic fact, and 24% of heavy smokers don't believe it.[55] Thus, these data indicate that millions of smokers do not know the basic fact that smoking is harmful to their health. However, these data present only a small part of the picture.

Even if every consumer were generally aware that smoking was somehow hazardous to health, the evidence indicates that a signficant percentage of consumers do not know enough about the specific health risks of smoking to assess their personal health risk from smoking or know *how dangerous* smoking is for them. The evidence indicates that many seriously *underestimate the severity* of the harms of smoking and the *probability* of suffering these harms. Many also do not know of the *existence of the relationship* between smoking and many of the most serious health consequences of smoking.

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[55] Gallup Opinion Index at 21; *see* Ch. III, *supra*.

It is relevant to a consumer's understanding of how dangerous smoking is, to know, for example, that it significantly reduces one's life expectancy, i.e., increases the risk of premature death. Yet, 30% of the population and 41% of smokers do not know that, for example, a 30 year old who smokes one pack a day, significantly reduces his life expectancy.[56] Among those who do know that smoking reduces one's life expectancy, many seriously underestimate the extent of the risk of premature death from smoking.[57] Moreover, 50%, or one-half, of light smokers falsely believe that only "heavy" smoking is hazardous.[58]

Despite the "overwhelming" medical evidence,[59] a large portion of the public still does not know smoking causes cancer. [Confidential Information Omitted] There is also substantial consumer ignorance concerning the relationship between smoking and specific cancers. Surveys indicate that between 19% and [Confidential Information Omittted] of the population, and between

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[56] 1980 Roper Study.

[57] *Id*.

[58] 1978 Roper, q. 16, 17; *see* Ch. III, Sec. II, *supra*.

[59] 1979 Surgeon General's Report, Ch. 1 at 16.

27% and [Confidential Information Omitted] of smokers, don't know smoking causes lung cancer,[60] while between one-quarter and one-half of the population is unaware that smoking is the major cause of lung cancer.[61] In addition, many more consumers seriously underestimate the risk of getting lung cancer as the result of smoking,[62] as well as the risk of dying from smoking related lung cancer.[63]

A substantial portion of the public also does not know about the relationship between smoking and several other serious health problems. Between 32% and [Confidential Information Omitted] of the population do not know that smoking causes heart disease, while among smokers between 37% and [Confidential Information Omitted] are unaware of this fact.[64] Over 50% of the population does not know that smoking causes many as opposed to just a few cases of heart attack.[65] About 10% of adults and 20% of teens do not know that smoking causes emphysema and bronchitis.[66] More than one-third of the population does not know that smoking causes many cases of emphysema and bronchitis, while over 60% do not know that smoking causes most cases of these diseases.[67] Large percentages of the population also do not know that maternal smoking during pregnancy threatens the life and health of the fetus. Over 40% of the population, approximately 50% of women and more than half of those who smoke,[68] don't know that maternal smoking increases the likelihood of miscarriage or stillbirth.[69] About two-thirds of college students polled, and three-quarters of the college student smokers, did not know about the increased risk of neonatal death from maternal smoking.[70]

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[60] The surveys were conducted by Gallup [Confidential Information Omitted]; see Ch. III, supra. See also 1980 Roper Study.

[61] 1978 Roper, q. 18h; see Ch. III, Sec. III, supra; 1980 Roper.

[62] Chilton, q. 20; see Ch. III, Sec. III, supra; 1980 Roper.

[63] Chilton, q. 24; see Ch. III, Sec. III, supra.

[64] See Ch. III, Sec. IV, supra.

[65] 1980 Roper.

[66] Chilton, q. 42(b)(c); see Ch. III, Sec. V, supra.

About one-half of the population does not know that smoking is considered addictive by many experts,[71] and a similar percentage does not know that cigarette smoke contains the toxic gas carbon monoxide.[72] In addition, the data suggest that substantial portions of the population overestimate the safety of low "tar" and nicotine cigarettes,[73] and underestimate the greatly increased risk of heart attack from smoking for women who take birth control pills. In fact, 36% of the population does not know that a woman who takes birth control pills substantially increases her risk of heart attack if she also smokes.[74]

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[67] 1980 Roper.

[68] Chilton, q. 39, 40.

[69] Chilton, q. 39, 40; see Ch. III, Sec. VI, supra; 1980 Roper. The Shor study, see Ch. III, Sec. VI, supra, found that about 75% of the college students sampled expressed doubt that smoking increases the risk of miscarriage or stillbirth.

[70] Shor, at 24; see Ch. III, Sec. VI., supra.

[71] 1978 Roper, q. 32; see Ch. III, Sec. VII, supra.

[72] Chilton, q. 43; see Ch. III, Sec. VII, supra.

[73] See Ch. III, Sec. VII, supra; 1980 Roper.

In conclusion, it appears that the "substantial portion" requirement is satisfied in this case. Viewed in its entirety, the evidence demonstrates that millions of Americans do not appreciate the impact of smoking on their life expectancy, their increased risk of suffering death or disease from cancer, heart attack and lung disease, or the serious danger to the fetus from maternal smoking during pregnancy.

2. Materiality of omitted facts.

A deceptive advertisement violates Section 5 if it fails to disclose information that is material or "likely to affect the average consumer in deciding whether to purchase the advertised product."[75] Traditionally, the Commission has not been required to conduct a consumer survey to determine whether the omitted information is material although such evidence is considered whenever available. "Once the Commission has found an advertisement to be deceptive, it is authorized within the bounds of reason, to infer that the deceptive information would be a material factor in the consumer's decision to buy."[76] In cases involving consumer health or safety, the Commission consistently has assumed that omitted health and safety information is material.[77] Thus, there is a presumption that information relevant to consumer health and safety is material.

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[74] See Ch. III, Sec. VII, supra; 1980 Roper.

[75] 1964 Cigarette Rule, supra at 8351.

The only health-related information provided by cigarette advertisers is the government-mandated health warning. As we have discussed in Chapter IV, Section II, while the current health warning represented a reasonable remedial effort nearly a decade ago based upon the evidence then available, the continued growth of medical knowledge about the health consequences of smoking and new evidence about the present ineffectiveness of the current warning indicates that a new informational remedy may now be necessary.

In analogous but not identical situations, the Commission previously has held that a general health warning may not be adequate and that specific information concerning the health hazards of smoking may be material. For example, in American Medicinal Products, Inc., et al., 32 F.T.C. 1376 (1941), aff'd, 136 F.2d 426 (9th Cir. 1943), advertised health warnings that were much more detailed than the current cigarette warning were found to be deceptive. In that case, it was held insufficient merely to warn that the preparation was not to be taken by children or by people suffering from tuberculosis, diabetes, or goiter, and that it should be used according to label directions. Instead, the seller was ordered to divulge that the preparation should only be used under medical supervision, that it was definitely harmful if used by persons suffering from certain specific diseases, and that it might cause breakdown of muscular tissue, irritation of nerve tissue, nervousness, irritability, and increased heart rate, with possible irreparable injury to the health of even a normal individual.[78] It should, nonetheless, be noted that these cases have not provided a conclusive formula for determining every situation in which advertisers will be required to provide more detailed health information than a general health warning. To date, the Commission has made its determinations of this issue based upon the facts of the particular case, including the nature and potential severity of the injury involved, a review of the advertisements in question and other facts it has found to be relevant.

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[76] Simeon Management Corp. v. F.T.C., 579 F.2d at 1146; See, e.g., F.T.C. v. Colgate Palmolive Co., 380 U.S. 374, 392 (1965).

[77] The Firestone Tire and Rubber Co., 81 F.T.C., 398, 451 (1972), aff'd, 481 F. 2d 246 (6th Cir. 1973), cert. denied, 414 U.S. 1112 (1973); Aronberg v. F.T.C., 132 F.2d 165, 168-69 (7th Cir. 1943); Simeon Management Corp. v. F.T.C., 579 F.2d at 1145; Seymour Dress and Blouse Co., 49 F.T.C. 1278, 1282 (1953); Academy Knitted Fabrics Corp., et al., 49 F.T.C. 697 (1952).

In the case of cigarette advertising, the health information omitted from the current ads is both necessary and material. First, the public lacks information about the specific hazards of smoking. Lacking this information, consumers cannot adequately assess the overall health risks of smoking in terms of the type of diseases they may develop, the severity of harm they might suffer or their increased likelihood of suffering these adverse health consequences. If any information is material to consumers, information that the product being advertised can kill them or lead to such diseases as cancer, heart attack, bronchitis or emphysema should fit this definition.

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[78] *See also*, *Positive Products Co.*, 33 F.T.C. 1327, *aff'd sub nom.* *Aronberg v. F.T.C.*, 132 F.2d 165 (7th Cir. 1942) (detailed health warnings required on ads for medicinal preparations); *Simeon Management Corp. v. F.T.C.*, 579 F.2d 1137 (9th Cir. 1978) (failure to disclose that drug lacked FDA approval for weight loss was deceptive).

Second, a substantial number of people say that they need more information about smoking and health and that they prefer a warning that discloses the specific dangers of smoking. Respondents were asked whether they knew enough about the issue of smoking and health or whether they needed to know more. One-quarter of both smokers and nonsmokers said they needed more information. Young people were more apt to want more information than older people. Of those who reported recently being exposed to any worrisome information about smoking, 40% felt that they needed more information.[79] These results substantiate previous HEW findings that half of the adult population "mildly disagree" or "strongly disagree" that "the general public knows all it needs to know about the effects of smoking and health."[80] Most important, nearly two of every three respondents judged the current warning inadequate and preferred a stronger, more specific warning listing particular dangers of smoking.[81]

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[79] 1978 Roper Report, q. 68.

[80] Department of Health, Education and Welfare, Center for Disease Control, National Cancer Institute, Adult Use of Tobacco, q. 3(b).

Third, people have considered health information in their smoking decisions. Ex-smokers cite "government reports on the effects of smoking and health" as an important reason they quit smoking.[82] More health information might also help the two-thirds of current smokers who say they want to quit smoking.[83] In addition to this self-report data, a recent study by the FTC Bureau of Economics[84]

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[81] 1978 Roper Report, q. 25. Sixty-one percent of all respondents (47% of smokers and 69% of nonsmokers) favored a warning described to them as "a specific one naming the hazards." It read, "Warning: cigarette smoking is dangerous to health, and may cause death from cancer, coronary heart disease, chronic bronchitis, pulmonary emphysema and other diseases." This is the same warning that the Commission recommended that Congress require on cigarette packages. F.T.C.'s Annual Report to Congress on Cigarette Advertising for the Year 1978 at 14.

[82] The 1978 Roper Report asked former smokers to choose from a list of items one or two factors that had the most to do with causing them to quit. The most often cited factor (37%) was "health problems." The third most cited factor (17%, compared with 14% in 1974) was government reports of the effects of smoking on health. Among young adults ages 21-24 "government reports" were cited as often as "health problems." Id., q. 61. [Confidential Information Omitted]

[83] Sixty-six percent of smokers would "sort of like to give it up," "very much like to quit," or "like to quit but can't." 1978 Roper, q. 43.

[84] F.T.C., Bureau of Economics, Staff Report on Consumer Responses to Cigarette Health Information, August, 1979. This report also concluded that while the televised public service announcements aired under the FCC's fairness doctine from 1968 to 1971 may have contributed to the decline in per capita cigarette consumption, there is no evidence that they had an effect unique from other information sources. This conclusion controverts those of prior studies and authorities that attribute a unique role in the decline to the PSAs. Warner, K., The Effects of the Anti-Smoking Campaign on Cigarette Consumption, Am. J. Public Health 645-650, (1979); Fishbein Study at

(Continued)

concludes that health information from a variety of sources, including the 1964 Surgeon General's Report, media publicity, warning labels, and televised public service announcements led to a gradual decline in per capita consumption of cigarettes from 1964 to 1975. Finally, the Surgeon General credits informational and educational public health campaigns of the past 15 years with "dramatic changes ... in adult smoking"[85] and urges officials to "ensure that smokers and potential smokers are adequately informed of the hazards."[86]

The preceding discussion indicates that in the staff's view, cigarette advertising has omitted material information about the health risks involved in cigarette smoking. Precisely what additional information should be disseminated to the public to enable it to accurately assess the risks of smoking should be evaluated after public comment and additional analysis.

C. The Possible Deception in Cigarette Advertising Would Seem Particularly Appropriate For Action Because Cigarettes Are Hazardous to Health, Adversely Affect Particularly Vulnerable Groups And Are The Subject of Continuous, Massive Advertising

The Commission has been particularly concerned about deceptive advertising when one or more of the following three factors is

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[84] (Footnote Continued)

67; Miller, R.H., Factors Affecting Cigarette Consumption, *U.S.D.A. Research Service* 1974; Hamilton, The Demand for Cigarettes: Advertising, the Health Scare, and the Cigarette Advertising Ban, *Rev. of Economics and Statistics* 401-11 (Nov. 1972); *Capital Broadcasting v. Mitchell*, 333 F. Supp. 582, 587 (D.D.C. 1971) (J. Skelly Wright dissenting), *aff'd*, 405 U.S. 1000 (1972).

[85] 1979 Surgeon General's Report, Ch. 19 at 9.

[86] *Id.*, Preface at xiv.

present: (1) the product is hazardous to health, (2) the product and/or the ad may adversely affect particularly vulnerable groups of consumers, and (3) any deception is aggravated by continuous, massive advertising. All three factors exist in cigarette advertising and would exacerbate its possible deception.

First, the deceptive advertising of products which affect consumer health and safety has often been the subject of Commission action. In these cases the Commission has required that all advertisements of such products be accurate and complete,[87] especially when the potential injury is severe and the number of people affected is large.[88] As the Commission held in Firestone Tire and Rubber Co,

> We note at the outset that both alleged misrepresentations go to the issue of the safety of respondent's product, an issue of great significance to consumers. On this issue the Commission has required scrupulous accuracy in advertising claims, for obvious reasons. If consumers are misled or uninformed as to the safety of a product, the consequences may not be limited to monetary loss but personal injury as well. Thus, the Commission has frequently decided that the omission of product safety information is an unfair and deceptive practice. 81 F.T.C. at 456, aff'd, 481 F.2d 246 (6th Cir. 1973), cert. denied, 414 U.S. 1112 (1973).

Second, advertisements which affect vulnerable consumers have also been subjected to particularly close scrutiny. In this case, this concern encompasses those who are subject to a high risk of personal injury from using an advertised product because of their physical condition or environment. This latter type of vulnerable audience includes those who have a physical condition that may be aggravated by use of a product,[89] such as those who are ill, pregnant,[90] obese,[91] and those who may for other reasons be particularly affected by use of the hazardous product.

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[87] National Commission on Egg Nutrition, et al., 88 F.T.C. 89, 162 (1976), modified, 570 F.2d 157 (7th Cir. 1977), cert. denied, 439 U.S. 821 (1978).

[88] 1964 Cigarette Rule, supra, at 8354.

Third, the Commission has recognized that a potentially deceptive advertisement cannot be viewed "in isolation," but must be considered "against the background" of the cumulative effect of years of similar advertising.[92] The total impact of prolonged and voluminous advertising may exacerbate the misimpression created by the advertising. Repeated exposure to a large number of ads that extol the virtues of a product without disclosing its specific negative consequences may cause consumers to ignore those negative facts.[93]

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[89] *American Medicinal Products, Inc. v. F.T.C.*, 136 F.2d 426 (9th Cir. 1943); *Miracle Hearing Aid, Inc.*, 49 F.T.C. 1410 (1953); *Pascal Co., Inc.*, 32 F.T.C. 1216 (1941).

[90] *Aronberg v. F.T.C.*, 132 F.2d 165 (7th Cir. 1943); *Dr. Jane Blanchard*, 32 F.T.C. 1291 (1941).

[91] *Simeon Management Co. v. F.T.C.*, 579 F.2d 1137 (9th Cir. 1978); *American Medicinal Products Inc., v. F.T.C.*, 136 F.2d 426 (9th Cir. 1943); *Porter & Dietsch, Inc., et al.*, 90 F.T.C. 770 (1977).

[92] *Warner-Lambert Co. v. F.T.C.*, 562 F.2d 749 (D.C. Cir. 1977); *see, Waltham Watch Co. v. F.T.C.*, 318 F.2d 28 (7th Cir. 1963), *cert. denied*, 375 U.S. 944 (1963); *Royal Baking Powder v. F.T.C.*, 281 F. 744 (2d Cir. 1922); 1964 Cigarette Rule, *supra*, at 8356-8357.

All three of these aggravating factors are applicable to current cigarette advertising. As discussed above, the scientific evidence that smoking is linked to various forms of cancer, heart disease and chronic obstructive lung disease is now overwhelming.

Cigarette advertising and smoking also affect certain vulnerable consumers. For example, pregnant women who smoke jeopardize the health and safety of the fetus and newborn infant. Women who take birth control pills greatly increase their risk of heart attack if they also smoke. Individuals who suffer from cardiovascular disease, chronic obstructive lung disease or anemia exacerbate their already existing health problems if they smoke.

Finally, cigarettes are the most heavily advertised product in America today. In 1979, two of the top five advertisers in magazines were cigarette companies. During the same year cigarettes were the most heavily advertised product in newspapers and almost one-half of all outdoor billboards in this country are devoted to cigarette advertising.

The applicability of all three factors to cigarette advertising makes it especially important that cigarette ads be accurate and complete in disclosing the health risks of smoking. Therefore, any deception in cigarette advertising practices may be particularly appropriate for action.

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[93] Id. See also The J.B. Williams Co. v. F.T.C., 381 F.2d, 884, 890 (6th Cir. 1967).

D. Conclusion

In sum, cigarette advertising does not disclose material health and safety information. As a result many smokers remain unaware of the very basic fact that smoking is hazardous to health and many more hold false beliefs about the existence, probability and severity of these dangers. In addition, the evidence indicates that the current warning is not an effective remedy for overcoming this problem.

CHAPTER V - REMEDIAL OPTIONS AND RECOMMENDATONS*

I. INTRODUCTION

The findings of this Report indicate that: (1) Over the past 15 years medical research has strengthened the evidence demonstrating the relationship of smoking to diseases such as cancer, heart attack and chronic obstructive lung disease and has established the relationship between smoking and a number of other health hazards about which little was known in 1964. The medical evidence accumulated over the past two decades also indicates that cigarette smoking is far more dangerous to health than was thought in 1964. (2) With the exception of the required health warning, cigarette advertising contains no health information. (3) While the number of Americans who are generally aware that smoking is in some way hazardous to health has grown steadily, some, especially smokers, still do not know this basic fact. More significantly, a much larger number know too little about the specific health hazards of smoking to be able to assess accurately how dangerous smoking is and whether the health risks of smoking have any personal relevance or application to themselves. (4) The current health warning is rarely noticed and is not effective in alerting consumers to the health hazards of smoking.

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* In addition to the information in this Chapter, the Commission relied upon information supplied by the cigarette companies pursuant to subpoena in a confidential marketing plan and in a confidential memo outlining a plan to bring to the public the industry's position on smoking and health. This information has been deleted from the version of the report released to the public.

Based upon these findings staff is concerned that current cigarette advertising practices may be deceptive. Staff has also tentatively concluded that additional action designed to provide consumers with more information about the health consequences of smoking is necessary. An effective remedy should be noticed by consumers, should maintain its noticeability over time, and should effectively convey sufficient information to prevent any possible deception.

In this investigation, staff has considered a number of remedial options, including (a) educational efforts of other governmental and private organizations, (b) voluntary industry self-regulation, (c) alteration of the size and shape of the warning, (d) replacement of the current warning with a single new, more specific warning, (e) replacement of the current warning with a rotational warning system, (f) tombstone advertising, and (g) disclosure of carbon monoxide levels. A number of these options are not mutually exclusive. For example, a change in the size and shape of the health warning can be accompanied by either the adoption of a new warning or a system of rotational warnings. A change in the warning can also be accompanied by increased educational efforts by other governmental and private organizations.

Many organizations, both inside and outside of government, have historically participated in efforts to inform the public of the health hazards of smoking. Congress has traditionally maintained exclusive jurisdiction over the warning on cigarette packages, while the Commission has taken responsibility for the health disclosures on cigarette advertisements. The Department of Health and Human

Services, along with various private organizations, has played the primary role for other educational efforts in this area. It should be emphasized that any option must be viewed as one part of an overall educational effort, if any option is to be effective. Therefore, to determine what action is ultimately most appropriate, as well as who should undertake that action, will require a cooperative effort on the part of Congress, the Commission and other appropriate governmental and private agencies.

II. REMEDIAL OPTIONS

A. Educational Efforts of Other Governmental and Private Organizations

Many believe that public information campaigns conducted through the mass media have the greatest impact on consumer knowledge. The classic example of such a campaign is the series of informational commercials on smoking that appeared on television between 1967 and 1971.[1] Although a recent study by the Commission's Bureau of Economics found that it is impossible to determine precisely the effectiveness of any single method of public education, including these public service announcements, the study also concludes that, as part of an overall educational effort, public service announcement campaigns do serve a useful purpose.[2] Therefore, staff has examined the role currently being played by public service campaigns sponsored by either governmental or private organizations in fulfilling the need to better inform consumers about the health hazards of smoking.

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[1] *See*, J. Hamilton, "The Demand for Cigarettes: Advertising, The Health Scare, and the Cigarette Advertising Ban," *Review of Economics and Statistics* 401-11 (Nov. 1972); Warner, "The Effects of the Anti-Smoking Campaign on Cigarette Consumption," 67 *American Journal of Public Health* 645-50 (July 1977). The effectiveness of these PSA's was judicially recognized by Judge Wright in his dissent in *Capitol Broadcasting Co. v. Mitchell*, 333 F. Supp. 582, 587 (D.D.C. 1971), *aff'd* 405 U.S. 1000 (1972).In referring to the Fairness Doctrine PSA's Judge Wright stated:

> For the first time in years, the statistics began to show a sustained trend toward lesser cigarette consumption...the anti-smoking messages were having a devastating effect on cigarette consumption. *Id*.

[2] F.T.C., Bureau of Economics, Staff Report on Consumer Responses to Cigarette Health Information, August 1979. The Bureau of Economics Report concludes that, although useful, the effect of the 1967-71 messages on smoking may have been overstated.

In the aftermath of the broadcast ban on cigarette commercials, the number of informational messages on smoking and health was also reduced. The present number and exposure of PSA's are inadequate to inform the public about the dangers of smoking and are not sufficient, by themselves, to remedy the problems discussed in this report. The primary government agency responsible for education on smoking is the Department of Health and Human Services' Office on Smoking and Health (OSH). OSH publishes smoking and health information, produces informational PSA's on smoking and health and serves as a clearinghouse for collecting and disseminating scientific and technical information on smoking and health. The entire OSH budget for Fiscal Year 1980 was $2.5 million.[3] Of that, only an estimated $750,000 was spent on public information.[4] The major expenditure was a $432,000 contract with an advertising agency to develop a media campaign.[5] The remainder of the public information funds were spent on the implementation of the agency's suggestions.

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[3] Letter from John Bagrosky of OSH to FTC (April 1, 1981).

[4] Id. In addition to the $750,000 spent on public information, an additional $500,000 was spent on technical information. Basically, the money was spent to compile a bibliography of smoking studies, to keep track of ongoing research, and to respond to requests for technical information from scientists, researchers, and the lay public.

[5] Id.

The effectiveness of mass media PSA's depends upon the frequency of their appearance and the size of the audience they reach. Between 1967 and 1971, smoking PSA's were seen often and by many people. Today, they are virtually invisible.

As part of its public education program, the Office on Smoking and Health (OSH) produces public service announcements (PSA's) on the health hazards of smoking. These PSA's are then broadcast without charge by the networks and by local television stations as part of their responsibility to broadcast in the public interest. OSH monitors how often and at what times these PSA's are broadcast.[6]

Two PSA's about smoking were in circulation in the Fall of 1980, but were infrequently shown, and even when shown, were seen at times when the fewest viewers watch television.

The three networks rarely broadcast the PSA's during prime time. During September and October 1980 only one was seen during prime time. The majority of the broadcasts were after midnight, with the remainder appearing very early in the morning, another light viewing time. Moreover, the data relied upon in monitoring indicates only when the network itself broadcast the PSA's. The network affiliate stations are under no obligation to include the PSAs broadcast by the network in their programming, so even these infrequently shown PSA's might not have been seen in every market.

The local television stations also do not broadcast many PSA's. In over 80% of the largest 75 markets during the monitored week in

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[6] The data in the following discussion of exposure of government PSA's comes from information provided by the Office on Smoking and Health.

September 1980, no government produced PSA about smoking was broadcast during prime time. Among the major markets without a prime time broadcast were Boston, Chicago, Detroit, Houston, Los Angeles, and Philadelphia. Of the PSA's broadcast at other times, most appeared during the daytime or late at night. Of the two messages monitored, one failed to reach fully 91.5% of the homes in America with TV's. In contrast, the six major cigarette manufacturers spent over one billion dollars to advertise their product in 1980.[7]

The government is not the only organization providing public education on smoking and health. Organizations such as the American Cancer Society, the American Heart Association, and the American Lung Association devote a percentage of their budgets to smoking education, including the production of public service announcements. Together, all of these organizations spend less than $10 million on their smoking-related public education efforts. For example, the American Cancer Society spends approximately $8 million annually on its efforts.[8] The American Lung Association spends nearly $1 million out of its $5.5 million budget on its public education campaign.[9] These are the largest private agencies providing public education. The American Heart Association has allocated $63,500 annually to develop its informational campaign on smoking and health, exclusive of production and dissemination costs.

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[7] See Chapter II, supra.

[8] Letter from Irving Rimer, Vice-President for Public Information for ACS, to FTC (July 17, 1980).

[9] 1978-79 American Lung Association Annual Report.

There is evidence that the current levels of public education efforts, particularly PSA's (both government and private), are ineffective. A 1976 Yankelovich study reported that only 49% of teenagers had seen at least one PSA on smoking and health within the previous month.[10] The 1978 Roper report conducted for the cigarette industry offers even more dramatic proof. Only 31% of the respondents had heard or read anything during the previous year that made them more or less worried about smoking. A mere 2% of that 31% reported that PSA's were responsible for their concern.[11] The most persuasive evidence that current educational efforts on smoking and health are inadequate is the investigation into the level of consumer knowledge detailed elsewhere in this report.[12]

In conclusion, the evidence indicates that at the present time public service announcements concerning smoking and health sponsored by both the government and private organizations appear infrequently and reach few consumers. To be effective these announcements need to appear more frequently during times when more consumers will see them.

Thus, the current public education efforts of the Office on Smoking and Health and other interested organizations are inadequate and cannot be relied upon to remedy the problems addressed by this report. Moreover, recent budget documents submitted to Congress propose a substantial reduction in the Office on Smoking and Health's budget, further reducing its ability to educate consumers about smoking-related issues. Nonetheless, public service announcements can play a useful role in informing consumers about the health hazards of smoking. Therefore, while an increase in the number of smoking-related public service announcements is not likely to alter the need for a change in the current health warning, an increase in these announcements as part of an overall educational effort is likely to result in some increase in the public's awareness of the health hazards of smoking.

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[10] Yankelovich, Shelley, & White Inc., Teenage Boys and Girls and Cigarette Smoking, a Supplemental, 40 (1976).

[11] 1978 Roper Report, q. 67.

[12] See Chapter III, *supra*.

B. Voluntary Self Regulation

The cigarette industry has not developed, and is not likely to develop, effective self-regulatory mechanisms to ensure that adequate health information is included in cigarette advertising. First, the cigarette manufacturers have never acknowledged the health hazards caused by smoking. Second, in the past the cigarette industry has agreed to take "voluntary" steps to better inform the public about the health hazards of smoking only after governmental action made it clear that the industry would be required to make these disclosures. Third, the cigarette manufacturers have not responded to concerns publicly expressed by members of Congress, by the Commission, and by concerned citizens, to limit certain aspects of cigarette advertising.

In 1964, when the Commission first considered requiring warning labels on cigarettes, self-regulation by the tobacco industry was advocated as an alternative to a Commission-mandated warning. However, the Commission found that "no industry representative has indicated that cigarette manufacturers are willing to disclose the health hazards of cigarette smoking in advertising...."[13] The willingness of the cigarette makers to disclose the dangers of smoking voluntarily appears to be no greater in 1981 than it was in 1964. Although the medical evidence on the hazards of smoking is now overwhelming, the industry continues publicly to refuse to recognize the validity of this evidence.[14] The unwillingness of the cigarette industry to acknowledge the dangers of smoking coupled with the industry's aggressive attacks on the validity of the scientific evidence counsels against relying on the industry to disclose the hazards at this time.[15]

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[13] 1964 Cigarette Rule at 8364.

The cigarette industry has also not successfully regulated itself in the past. For example, unlike the liquor industry, cigarette manufacturers have never produced an advertisement discouraging use of their product by young people and children.[16]

The few instances of voluntary self-regulation on the part of the cigarette industry have been prompted by the existence of governmental proposals to make these actions mandatory. For example, the major cigarette manufacturers "agreed" to disclose "tar" and nicotine levels in their advertisements only after the Commission proposed a Trade Regulation Rule in 1970 that would have made it an unfair or deceptive practice to fail to do so.[17] The Commission's proposal did not cover the disclosure of "tar" and nicotine content on cigarette packages because Congress, in the Federal Cigarette Labeling and Advertising Act,[18] had retained jurisdiction over all health disclosures on cigarette packages.[19] Consequently, a decade later most cigarette packages still do not contain this information. Similarly, despite the Congressional requirement that all cigarette packages contain a health warning, the cigarette industry did not agree to include the same warning in its advertisments until after the Commission announced its intention to file complaints against each of the cigarette companies, alleging that advertisements without a clear and conspicuous health disclosure were "false and misleading" and constituted an "unfair practice."[20] Subsequently, problems have developed in the implementation of this agreement. In 1975, the Commission voted to seek civil penalties against the major cigarette manufacturers for violating these consent orders.[21]

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[14] See Ch. I, Section II, J., *supra*. [Confidential Information Omitted]

[15] [Confidential Information Omitted]

[16] Blum, *Commentary: Medicine vs. Madison Avenue*, 243 Journal of the American Medical Association 739 (1980).

[17] See Ch. IV, Sec. I.

[18] 15 U.S.C. §§ 1331 *et seq*. (1970).

The industry's only experiment with self-regulation, the Cigarette Advertiser's Code, which regulated broadcast advertising, was established in 1964 in response to the Commission's 1964 rulemaking action and ceased operation in August 1970 shortly before the congressionally-mandated broadcast ban went into effect. While some members of the tobacco industry assert that they still adhere to the principles of the code,[22] a review of current advertising practices[23] indicates that the code has little, if any, practical effect.

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[19] See Ch. IV, Sec. I.

[20] _Id._

[21] _Id._ at n. 14.

[22] _See, e.g._, Statement of the Tobacco Institute Re S. 3118 submitted to the Subcommittee on Health and Scientific Research of the Committee on Human Resources, United States Senate, July 12, 1978.

The major provisions of the 1964 Cigarette Advertisers Code limited advertising on Radio and TV to programs not "primarily" directed at children, required models to be at least 25 years old, prohibited endorsements by athletes or the association of smoking with athletic activities, and prohibited any health claims in relation to cigarettes.[24]

Problems developed with the administration of the Code from the outset. Two of the major cigarette companies, Lorillard and American Tobacco, withdrew at an early stage.[25] From the beginning the Code was also criticized for its ineffectiveness in keeping cigarette advertisements away from children.[26] As a result, the industry did agree to restrict its ads to shows where the audience under the age of 21 did not exceed 45%.[27] Even with this loosely enforced attempt, however, many children continued to be exposed to cigarette advertising.

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[23] See Ch. II.

[24] What's Ahead for the Code, Tobacco Reporter, November 1967 at 18.

[25] Lorillard withdrew in March 1966 immediately after the Commission announced that "tar" and nicotine figures could be mentioned in cigarette ads, a practice not permitted by the Code. American Tobacco withdrew in September 1967 because of the promulgation of the National Association of Broadcasters Code.

[26] Advertising Age, Sept. 27, 1965, at 1.

[27] Advertising Age, May 9, 1966, at 1.

In 1969, the Commission evaluated the Code's effectiveness. While praising some of the Code's successes, such as removing athletes from commercials,[28] the Commission's ultimate judgment on the Code's effectiveness was negative. The 1969 Report to Congress stated:

> Whatever the policy of the NAB, the Code Authority or the CAC since April 16, 1968, the current Commission review of cigarette advertising themes as well as reviews conducted in 1964, 1967, and 1968 amply demonstrate the futility in relying upon voluntary regulation of cigarette advertising to achieve any significant changes in the content and meaning of cigarette advertising.[29]

The analysis of the current themes of cigarette advertising in this report demonstrates that little has changed in the last 11 years.

The cigarette manufacturers have also failed to respond to suggestions that more useful health information be put into cigarette advertisements. The Commission, in several of its annual reports to Congress, has made recommendations for increasing the dissemination of health information on cigarettes to the public. For example, in the 1978 report, the Commission recommended to Congress that new specific warnings replace the current warning, and that "tar" and nicotine figures be required on each package of cigarettes. The cigarette industry never responded.

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[28] F.T.C.'s Annual Report to Congress on Cigarette Advertising for the Year 1969 at 27.

[29] Id. at 30.

The National Commission on Smoking and Public Policy in its 1978 report to the American Cancer Society recommended: 1) eliminating models in cigarette advertising; 2) limiting advertising to low "tar" cigarettes; 3) including "tar" and nicotine figures on each pack; 4) ensuring that the present health warning is visible, especially on billboards, and 5) refraining from any promotion aimed at anyone under 19.[30] The report concluded:

> We believe that government, voluntary agencies and the tobacco industry should work together to discourage young people from starting to smoke. Industry spokesmen say they are willing to promote cigarettes as an "adult custom." We believe that there is danger in such promotion: too many youngsters imitate adults. If there can be agreement in principle, however, that the aim is to discourage smoking among young people, cooperation to this end should be sought.[31]

The cigarette industry has not adopted any of these proposals.

In 1979, 38 Congressmen wrote to the Commission, urging it to negotiate a self-regulation system with the cigarette manufacturers.[32] The Congressmen advocated setting up a voluntary code "prohibiting the depiction of scenes or copy implying that smoking cigarettes leads to youth, sex, manliness, femininity, courage, or glamour, [which] would be a decisive step in eliminating the exploitation of young people who are especially vulnerable to claims of this type."[33] The industry responded by denying that its advertising practices had any relation to smoking among youths. Congressman Don Edwards pursued this issue, and offered the Tobacco Institute several examples of what he considered offensive advertisements directed at young people.[34] Once again, the industry took no action.

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[30] A National Dilemma: Cigarette Smoking or the Health of Americans, Report of the National Commission on Smoking and Public Policy, 1978, at 18, 51.

[31] Id. at 75.

[32] Letter from the Honorable Don Edwards and 37 other Members of Congress to Chairman, Federal Trade Commission (February 1, 1979).

It is particularly unlikely that the cigarette industry will voluntarily adopt a system of rotational warnings. In testimony in 1978 on proposed legislation regulating tobacco, the Tobacco Institute spoke out against rotational warnings. The Institute had three concerns. First, it felt that specific warnings would be misleading and counterproductive. Second, it argued that there is no need for additional information. Third, it feared that collecting the warnings could become a teen hobby.

In light of the cigarette industry's position that smoking does not pose a hazard to health, that its current advertising practices do not pose any problems, and that the public is already well-informed, combined with the industry's failure to regulate itself effectively in the past, voluntary industry self-regulation does not appear to be a reasonable alternative as a remedy to the current problems in cigarette advertisements.

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[33] Id.

[34] Letter from Honorable Don Edwards to Horace R. Kornegay, President, Tobacco Institute (August 30, 1979).

C. Alteration of the Shape and Size of the Warning

One of the major deficiences of the current warning is that very few people notice or read it. A contributing factor to this problem is the fact that for the past nine years the current warning has appeared in cigarette advertisements in the same, small rectangle.[35] Therefore, as part of this investigation staff examined whether altering the shape and size of the warning format would improve its noticeability and effectiveness. After a careful examination of the available evidence staff believes that a change in both the size and basic shape of the warning symbol would substantially improve its effectiveness. However, it should also be stressed that, although a change in the shape and an increase in the size of the current warning would increase its noticeability, these changes would not eliminate the need to communicate additional information about the health consequences of smoking to consumers. This remedial option, therefore, should be considered in addition to, and not instead of, any of the informational remedies discussed.

1. Shape of the Warning

To test whether altering the shape of the warning would improve its noticeability and effectiveness, staff commissioned Burke Marketing Research, Inc.[36] to conduct a "focus group" study[37] to gauge consumer reactions and attitudes towards several proposed shapes. The shapes were developed by Keenan and McLaughlin, a New York advertising agency. Of the nine shapes tested, two -- an eight-sided figure (octagon) half black and half white, and an arrow pointing into a circle -- emerged as the most likely to be noticed and understood.[38]

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[35] See, Ch. IV, Sec. II.

[36] Burke, Exploratory Print Focus Groups, January, 1980.

These two shapes were then further tested against each other and the currently used rectangle in a Portfolio Study conducted by Burke in June and July, 1980.[39] As part of this study interviews were conducted in shopping malls in six different cities across the nation. Overall, 1228 respondents were tested for aided and unaided recall of ads containing three different warnings[40] in four shapes:

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[37] The focus groups were drawn from groups in the population from whom we sought answers to our questions about the relative effectiveness of various shapes, i.e., smokers and nonsmokers and persons of varying age and sex. There was no representative selection of these people, so no statistical inferences can be drawn. Focus groups are, however, a commonly used technique in marketing research. They are a series of small-group discussions with a trained moderator designed to gather information to clarify existing theories, redirect efforts away from previous expectations, generate new ideas, and give direction to future research.

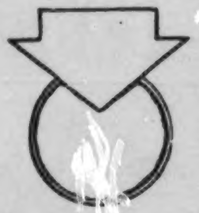
[38] The nine tested formats included the following: 1) an octagon that was half black and half white; 2) an all white octagon; 3) a white octagon with a band of black across the middle; 4) an arrow pointing into a circle; 5) a plain circle; 6) a circle surrounded by a square; 7) a circle that was half black and half white; 8) a square; and 9) a rectangle. See Appendix B for illustrations.

[39] Burke, Cigarette Print Ad Portfolio Study, November 1980 (Burke Portfolio Study).

[40] The warnings included the present Surgeon General's warning and two additional ones: "Lung Cancer: The Major Cause Is Smoking," and "Heart Attack: A Major Risk Factor Is Smoking."

the octagon, the circle and arrow, the current rectangle, and an enlarged rectangle.

Burke concluded that, overall, the circle and arrow and the octagon were noticed more often,[41] and yielded significantly greater unaided recall than the rectangle shape.[42] Total recall, both aided and unaided, of each of the warning messages was highest for the three warnings tested in the circle and arrow.[43] The company reported that the circle and arrow was superior to all the other shapes, including the octagon, in prompting mentions of the contents of the warning messages.[44] In sum, the circle and arrow shape was superior to all other shapes tested.[45] The currently used rectangle, on the other hand, consistently scored the lowest in terms of noticeability and recall.[46] Thus, staff recommends using the following circle and arrow format for the proposed rotational warnings:



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[41] Burke Portfolio Study at 1.

[42] Id., at 2. Unaided recall of a new lung cancer message in the circle and arrow reached 64%, while unaided recall of the present warning, which has been on packages since 1969 and in advertising since 1972, in its existing shape, was only 28%.

[43] Id., at 8, 12.

[44] Id., at 4.

[45] Id., at 2, 6, 7, 8.

[46] Id, at 4, 6, 8.

2. Size of the Warning

Part of the reason very few people notice the current warning is its small size. The other shapes tested by Burke, which were 50% larger than the existing rectangle, were all found to be more noticeable. A heart attack warning presented in the current rectangle was recalled, unaided, by only 33% of respondents,[47] one of the lowest showings of any format tested. By contrast, the same message when tested in a somewhat larger rectangle was recalled, unaided, by 38% of respondents.[48] The nonrectangular shapes, both of which were 50% larger in area than the present rectangle, scored significantly higher in every catagory than the present rectangle. For example, the same heart attack message that was recalled unaided by only 33% of those tested when presented in the current rectangle was recalled, unaided, by 57% when presented in the circle and arrow.[49]

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[47] Id., at 2. The current warning in the current rectangle had an unaided recall score of only 28%, the lowest score. The 28% score cannot be compared with the noticeability score obtained from the Starch test results, supra, Ch. IV, Sec. II. While Starch tested actual readership of ads by consumers when not in a test-taking situation, Burke asked participants to review the ads in question as part of a test of recall of the ad in which the participant had agreed to participate before reviewing the ad.

[48] Id.

[49] Id., at 4, 6, 8. Tests were conducted for unaided recall, total (aided) recall, first recall, and recall of the specific content of the warnings tested. Several warnings were tested in each shape. Regardless of the nature of the recall being tested, or the content of the warning, the larger non-rectangular shapes tested better than the current rectangle.

Preliminary data, therefore, seem to indicate that an increase in the size of the health warning would improve its noticeability. At this point staff makes no recommendation as to the precise dimensions of a new warning symbol. Additional research and public comment is needed to clarify exactly what size offers maximum increased effectiveness without imposing undue costs or taking up an undue amount of space. The warning should be large enough to be noticed, but small enough to permit the advertiser to communicate its desired message.

The only potentially significant cost of the proposed change is the cost to the manufacturer for the increased advertising space occupied by a warning if it is larger than the current warning. The use of this space, which might otherwise be used for the advertisement message, may be considered as imposing a cost on the manufacturer. Until the precise size of any new warning is selected, it is impossible to compute the exact cost of any increase in size. It should be noted that the cost is not necessarily proportional to the amount of space used in the ad and that the computation of the costs would be affected by a number of factors, such as the degree of flexibility permitted the advertiser to choose the symbol's location within the ad or whether the warning occupies space needed to communicate the advertiser's commercial message. Of course, as long as a new warning is not so large as to require manufacturers to buy bigger ads to communicate the same message, there is no increased out of pocket expenditure to the cigarette manufacturers. In addition, as long as the new warning continues to occupy only a small percentage of the space in print media, sidewalk, transit and point of purchase ads and does not occupy space needed to communicate the manufacturers' message, the increased cost in terms of lost space to the manufacturers is not likely to be substantial.

D. Single New Warning

One option to remedy the problems described in this report is to replace the current warning with a single, more detailed warning. For nearly a decade the Commission has been advocating that the current warning should be replaced by a more detailed, more specific health warning. In every Report to Congress filed by the Commission since 1972 pursuant to the Federal Cigarette Labeling and Advertising Act, the Commission has recommended that the current warning be replaced by the following message:

> WARNING: CIGARETTE SMOKING IS DANGEROUS TO HEALTH, AND MAY CAUSE DEATH FROM CANCER, CORONARY HEART DISEASE, CHRONIC BRONCHITIS, PULMONARY EMPHYSEMA AND OTHER DISEASES.[50]

In its 1976 and 1977 Reports, the Commission also informed Congress that the American Cancer Society's Board of Directors had approved a recommendation that the warning on cigarette packages and advertisements be amended to read:

> WARNING: CIGARETTE SMOKING IS A MAJOR HEALTH HAZARD AND MAY RESULT IN YOUR DEATH.

The Commission added that it believed that either of these warning statements "would constitute a desirable improvement in the warning being given to consumers."[51]

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[50] See, 1972, 1973, 1974, 1975, 1976, 1977 and 1978 FTC Reports to Congress pursuant to the Federal Cigarette Labeling and Advertising Act. See Appendix E for a sample of this warning in an advertisement format.

[51] 1976, 1977 FTC Reports to Congress pursuant to the Federal Cigarette Labeling and Advertising Act.

There are a number of advantages to replacing the current warning with a new, more specific warning. First, consumers have indicated that they prefer a more detailed warning. In the 1978 Roper Study conducted for the Tobacco Institute, consumers were asked whether they favored a warning identical to the one proposed by the Commission in its 1978 Report to Congress or the current warning. Sixty-one percent of those polled and 69% of the non-smokers polled favored the proposed new warning.[52] Only 34% of those polled and 26% of the non-smokers favored the current warning.[53]

Second, a new warning identical or similar to the warning the Commission has recommended to Congress in the past would be both more concrete and more informative than the current warning. Not only does the warning the Commission proposed inform consumers about the relationship between smoking and cancer, heart disease, chronic bronchitis and emphysema, it informs consumers that they can die from these smoking-related illnesses. Each of these facts provides consumers with additional information needed to evaluate the overall health risks of smoking. In addition, as was noted in Chapter IV, one of the factors contributing to the current warning's ineffectiveness is its abstract and general nature. The inclusion of the names of four of the specific smoking-related diseases and the fact that there is a risk of dying from these diseases makes the warning both more specific and more concrete.

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[52] 1978 Roper, q. 25.

[53] *Id.*

The third advantage to replacing the current warning with a new, more informative warning would be derived from the very fact that for at least some period of time, it would be perceived as being new. The fact that the old warning has not been changed for a decade has contributed to its ineffectiveness.[54] It is "worn out" and no longer communicates any new information to most consumers. Therefore, any change in the current warning, especially one that communicates new information to consumers, is likely to improve on the warning's noticeability and effectiveness for some period of time.

Thus, as the Commission has recognized since 1972, a single new more specific warning has numerous advantages over the current warning. It also appears that the current warning could be replaced with a single new warning without substantial cost or additional administrative burden to cigarette manufacturers or their advertising agencies. As long as the timing of the implementation of the new warning is coordinated with the cigarette industry's introduction of new advertising, replacement of the current warning with a new single warning would not add substantial additional production or administrative costs. Therefore, if the current warning were replaced, cigarette manufacturers should be given adequate prior notice to permit them to include the new warning in any advertising campaign scheduled to begin before and run beyond the formal implementation date in order to minimize the costs involved.

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[54] See Chapter IV, Section II. It should also be reemphasized that a change in the shape and size of the warning format, as recommended above, would increase the novelty and improve the effectiveness of a new warning message.

While there are numerous advantages to a single, new, more specific warning over the current warning, the merits of this change also need to be compared with the merits of replacing the current warning with the system of rotational warnings discussed subsequently in this report. When compared with a system of rotational warnings, a single new warning has three major drawbacks. First, any single warning is capable of effectively communicating only a limited amount of information. Although the warning proposed by the Commission in its annual reports to Congress is longer than the current warning, it, too, omits a substantial amount of significant, material health information. For example, the proposed warning contains no information about the risks of smoking by pregnant women or the increased dangers of smoking for women who also take birth control pills, or for individuals with pre-existing medical conditions which are aggravated by smoking. It does not inform consumers that low "tar" and nicotine cigarettes have not been proven to be safe or that smoking less than a pack of cigarettes each day is dangerous. The proposed warning also does not mention that many experts have concluded that smoking is addictive, a particularly important fact to a non-smoking consumer deciding whether to experiment with cigarettes, or that cigarette smoke contains carbon monoxide and numerous additives, some of which are known carcinogens. Similarly, the proposed warning does not indicate whether the increased risk of heart attack, cancer, emphysema or chronic bronchitis is large or small, nor does it indicate whether the risk of death from these smoking-related diseases is substantial or insignificant. In contrast, a system of specific rotational warnings is capable of communicating as much health information as the Commission or Congress deems appropriate and necessary.

The second drawback to a single new warning when compared to a system of rotational warnings relates to the length of the warning message. The current warning is 14 words long. The warning previously proposed by the Commission is 23 words long. If the current use of the name of the Surgeon General is retained in the proposed warning, the warning would be 28 words long. If the proposed warning were redrafted to include additional health information, its length would increase further, and even then, it is unlikely that a single warning could be drafted which would include substantially more health information than the proposed warning.

Increasing the length of the health warning has several implications. A longer warning would take up more space in the ad. On billboards this poses a particularly serious problem. In 1975, the Commission filed a case seeking civil penalties against the six major cigarette manufacturers. The complaint was amended in 1976 to allege that the size of the lettering of the current warning in billboards violated the 1972 consent agreement which requires that the warning be "clear and conspicuous." U.S. v. R.J. Reynolds Tobacco Company, 76 Civ. 813 (S.D.N.Y.). In this still pending action, the Commission has urged that to be "clear and conspicuous" to the average viewer of a billboard from the distance at which the billboard is normally seen, the size of the letters of the warning needs to be substantially increased. The average billboard is viewed at distances ranging from 100 to 400 feet. The Institute of Outdoor

Advertising states that all outdoor advertisements should be designed so that they will be legible at distances up to 400 feet.[55] Thus, to be legible on a billboard, a warning approximately twice as long as the current warning would have to occupy a substantial amount of a billboard's advertising space.

The length of the warning also has an impact on its effectiveness. Informational disclosures, especially on billboards, need to be brief, simple and concise to be most effective.[56] The Commission's recent task force on Consumer Information Remedies concluded, "when providing or requiring information disclosures, it should be remembered that consumers generally have limited processing capacities, which preclude them from being able to use 'too much' information."[57] Therefore, the task force concluded that in drafting an informational remedy, caution must be taken to avoid overloading consumers' short term memory.[58] Two recent studies conducted for the F.T.C. support this conclusion.

In a 1980 focus group interview study conducted by Burke Marketing Research, Inc. to evaluate the effectiveness of a number of different cigarette health warnings and warning formats, the researchers concluded that complex warnings that contained more than one idea were more difficult for consumers to understand.[59] In a second study, Walker Research, Inc. evaluated three variations of twelve different health warnings to determine which of the three variations was easiest to understand and which provoked the most thought about the health effects mentioned. While the study was not explicitly designed to test the impact of the length of a health warning on its effectivness, a review of the results indicates that the more simple and more direct warnings consistently scored better.[60] Among the warnings tested by Walker was the warning previously proposed by the Commission in its reports to Congress. This warning was tested against the following two shorter warnings:

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[55] Institute of Outdoor Advertising, A Creative Guide to Outdoor Advertising, at 6, 14.

[56] Id.; F.T.C., Consumer Information Remedies: Policy Review Session (June 1979).

[57] Id. at 103.

[58] Id.

> "Smoking Causes Cancer of the Lungs,
> Larynx, Mouth and Esophagus"
>
> "Cigarette Smoking Can Cause Death From
> Cancer, Heart Disease or Lung Disease."

Both of these warnings scored better than the warning previously proposed by the Commission in both understandability and ability to provoke thought about the health effects of smoking. In each case the warning which read, "Cigarette Smoking Can Cause Death From Cancer, Heart Disease or Lung Cancer," scored significantly higher than the longer warning previously proposed by the Commission.

The third drawback to a single new warning is that it would have many of the same problems which are responsible for the ineffectiveness of the current warning. Once in place, the new warning would not be changed. Therefore, it would become "overexposed" and "wornout" much more rapidly than would warnings included in a rotational warning system. The novelty of the new warning would also wearout more quickly than would rotational warnings. The decrease in novelty would make it less noticeable, less memorable, and less likely to be used by consumers in making smoking decisions. Concerns about keeping the warning short enough to be understood may also affect how concrete the message can be. Rather than disseminating the data on the hazards of smoking through a series of short, concrete, easily personalizable messages, a new single warning might be more general. In so doing, it may become more abstract, and therefore, less effective.

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[59] Burke Marketing Research, Inc., Exploratory Print Focus Groups (January 1980), at 6, 11.

[60] Walker Research, Inc., Health Warning Statement Concept Evaluation, (November 1980).

Finally, a preliminary investigation of the relative cost of a single new warning versus a system of rotational warnings indicates that there may not be a significant difference. When the rotational warning and the ad are produced at the same time, and no changes in the warning need be made, the ad should cost no more to produce than it would at the present time. An increase in production cost should also be incurred only when a particular ad runs for longer than the time specified for the particular warning. The difference in the administrative costs between the operation of a reasonable rotational system and a single new warning should also not be substantial. Preliminary research also indicates that once the ad has been produced, and the proper warning message inserted, the primary added cost should be the additional time it takes to monitor the schedule to ensure the correct warning is in the advertisement.

E. Rotational Warnings

Although a change in the shape and an increase in the size of the current warning, as proposed previously in this Chapter, would temporarily increase its noticeability, these changes would not by themselves remedy the problems in cigarette advertising. The current warning is worn out and provides no specific health information. Therefore, the content of the current warning needs to be changed. As was previously noted, a single new warning, even if more specific, would not adequately cure the problem caused by the public's gap in knowledge, nor would it overcome all of the underlying causes of the current warning's ineffectiveness.

Therefore, staff has evaluated the merits of a rotational warning system containing a number of short, specific, easily understandable warnings which would rotate among cigarette ads on a regular basis, perhaps quarterly. A system of rotated health disclosures would make it possible to provide consumers with sufficient health information to assess the risks of smoking and determine whether these health risks have any personal relevance to themselves. In addition, rotation of the warning would help eliminate the underlying causes of the present warning's failures.[61]

First, a rotational warning system would provide sufficient repetition of each message to contribute to long term recall of that message, while decreasing the likelihood that any one message would become so familar and so overexposed that its effectiveness would

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[61] See Ch. IV, Sec. II, supra.

"wear out."[62] Second, the use of a number of different, specific warnings that are rotated regularly would assist in maintaining the novelty of each warning. Moreover, the use of a number of different warnings, which would vary from brand to brand at any one time and which would regularly change, would make it more likely that consumers would consider the content of the warning message because novel information is not only more noticeable, it is more likely to be spontaneously recalled.[63] Third, the warnings in a rotational warning system would be shorter, more specific and more concrete than the current warning. It has been shown that concrete information is more easily transformed into mental images by consumers and is better remembered than abstract information. Studies have also shown that people rely on concrete information more than on abstract information.[64] Fourth, information which consumers perceive as having personal relevance is more likely to be noticed and remembered. The evidence indicates that information which is specific and concrete is more likely to be perceived as being personally relevant.[65] Therefore, a series of concrete, personalized rotating warnings would be more likely to be accessible, to be spontaneoulsly recalled, and to become part of consumer decision-making than the present warning.

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[62] See, Craig et al., Advertising Wearout: An Experimental Analysis, J. of Marketing Research (Nov. 1976).

[63] Cohen & Srull, Information Processing Issues Involved in the Communication and Retrieval of Cigarette Warning Information, Report prepared for the Federal Trade Commission, November 1980 at 12.

[64] Id., at 20.

[65] Rodgers, Kuiper & Kirker, Self Reference and the Encoding of Personal Information, 35 Journal of Personality and Social Psychology, 677-88 (1977).

The concept of rotational health warnings in cigarette advertising is not new. The Swedish government instituted a system of rotational health warnings in early 1977.[66] While the Swedish system's long-term impact cannot yet be measured, some preliminary research conducted by the Swedish National Smoking and Health Association led that organization to conclude that the new system has increased consumer knowledge and has had a beneficial impact on smoking behavior in that country.[67]

1. Content of the Health Warnings

The health warnings ultimately selected should be evaluated according to four criteria. They should be medically accurate, fill a demonstrable gap in consumer knowledge about the health hazards of smoking, be understandable, and prompt consumers to think about the health hazards of smoking.

The sample warnings suggested by the staff have been evaluated for their accuracy by the leading medical experts in the particular field of expertise. Each is supported directly by statements and conclusions of the 1979, 1980 and 1981 Surgeon General's Reports, or by the underlying studies relied on in those reports. Furthermore, each warning concerns a serious health hazard affecting a substantial portion of the population.

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[66] The Swedes rotate sixteen warnings. See Appendix C.

[67] Staff recognizes that there are a number of differences between the U.S. and Sweden that make it impossible to state that their experience with rotational warnings would be comparable to ours. Nonetheless, their experience is instructive.

Each sample warning also provides information about the health hazards of smoking about which there is a significant, demonstrable gap in consumer knowledge. The suggested new warnings, therefore, were tailored to the results of this report's assessment of the level of consumer knowledge about smoking.[68]

Staff also recognized the need to present the information in a manner that is both understandable and that prompts consumers to consider the health effects of smoking. Therefore, Walker Research, Inc., was commissioned to help develop sample warnings that met these two criteria. Walker conducted a nationally projectable shopping center intercept survey, in August and September 1980, in three cities in different parts of the country. Overall, 805 persons were asked to evaluate 36 proposed warnings -- 3 different warnings for each of 12 medical "concept" or fact areas -- as to their "understandability" and "ability to prompt people to think about the health effects of smoking."[69]

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[68] See Ch. III, supra.

[69] Walker Research, Inc., Health Warning Concept Study, Nov. 1980. Respondents were asked to rank the three proposed warnings in each concept area in terms of which made them "think most about the health effects of smoking." Respondents were also asked to rank the warnings in each concept area as to how easy they were to understand. In addition, respondents were asked to explain why they ranked the concepts the way they did, and their explanations were recorded verbatim.

Based in part on the Walker results and after careful consideration of all of the four criteria explained above, warnings along the following lines are suggested as illustrative of the types of warnings the Commission might consider.[70] It should be emphasized that the following have not all been thoroughly tested and are offered solely as illustrations of the type of warnings which might be included in a system of rotational warnings. (The capitalized words should appear in the arrow section of the shape, with the rest of the warning in the circle. See Appendix E for illustration of sample warnings in an advertisement format.)

a. WARNING: Smoking causes death from cancer, heart attack and lung disease.

b. LUNG CANCER: Smoking is the major cause.

c. LUNG CANCER: Most of its victims die within one year.

d. LUNG CANCER: 8 out of 10 cases are caused by by smoking.

e. HEART ATTACK: The number one cause of death from smoking.

f. HEART ATTACK: Smoking is a major cause.

g. CHRONIC BRONCHITIS AND EMPHYSEMA: Most cases are caused by smoking.

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[70] For several major subject areas, multiple warnings are suggested. In many subject areas, it is important that more than one warning be provided, although it is unlikely that all of the warnings suggested for a particular subject will be included. The precise content and number of warnings should be properly determined only after analysis of public comments and additional data developed in the future.

h. LUNG DISEASE:	The more you smoke, the more likely you are to die from chronic bronchitis or emphysema.
i. PREGNANT WOMEN:	Smoking increases the risk of spontaneous abortion and stillbirth.
j. PREGNANT WOMEN:	Smoking increases the risk of death of your unborn child.
k. WOMEN:	If you take the "pill", smoking greatly increases your risk of heart attack.
l. CARBON MONOXIDE:	Cigarette smoke contains carbon monoxide and other poison gases.
m. TEENS:	The earlier you start, the more likely you are to die from smoking.
n. WARNING:	Smoking may be addictive.
o. LIGHT SMOKERS:	Even a few cigarettes a day are dangerous.
p. SMOKERS:	No matter how long you have smoked, quitting greatly reduces your health risks.

The expense of the rotational warning system remains approximately the same whether three, four, or sixteen health warnings are required.[71] The primary cost that increases as additional warnings are required is the minor production cost associated with printing the additional warning.

To evaluate the costs and burdens of a rotational warning system, experts from an advertising agency have been consulted about how such a system could operate. Among the issues they have addressed include: (1) How frequently should the warnings be rotated in light of current advertising practices to provide for adequate exposure for each warning at a reasonable cost? (2) Should all brands carry the same warning at the same time or should warnings be rotated on a brand-by-brand or company-by-company basis? (3) How would the system operate in the print media, on sidewalk posters and transit placards, on give-away items and point-of-purchase advertising material, and on outdoor billboards? A discussion of how a rotational warning system might operate based upon the staff's preliminary investigation into this issue is included in Appendix D.

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[71] Keenan & McLaughlin, Inc., Cigarette Warning Project, 1981.

2. Costs Of Implementation Of Rotational Warning System

Staff has, with the consultation of our advertising agency, attempted to design the rotational warning system to keep costs to a minimum. To reduce the costs of a rotational system, its implementation should be coordinated with the cigarette industry's introduction of new advertising. In this way, the advertisers could add the new warnings to advertisements in the preproduction stage, rather than replace the current warning in an existing ad.

Most of the cost of the proposed system would be in the actual production of the ad. When the warning and the ad are produced at the same time, and no changes in the warning need be made, the ad should not cost more to produce than it would at the present time. The increase in production cost would be incurred only when a particular ad runs for more than the specified period selected and the warning must be replaced. If an ad overlaps the specified period, it appears that changing the warning in the production material of one 4-color magazine ad would cost approximately between $40-$245; changing a newspaper ad would cost approximately $150.

These costs are not substantial given the total cost of producing an advertisement. By rotating messages quarterly, rather than monthly as was once considered, the increased production costs should also be reduced.

Preliminary research also indicates that the administrative costs of operating the proposed system should not be unduly large. Most of the non-production costs of advertising appear to come in the initiation and creation of an ad campaign. The rotational system should not have a major effect on these costs. Once the ad has been produced, and the proper warning message inserted, the primary added cost should be the additional time it takes to monitor the schedule to ensure the correct warning is in the advertisement. This monitoring should not involve substantial time. The advertising agencies' traffic manager already prepares "schedule flow charts" indicating what advertisement runs in what publication. The primary administrative task imposed by the rotational system would appear to be that the traffic manager would have to keep track of the rotation schedule to see whether the proper warning had been inserted in each ad. If the ad does not overlap rotational periods in any publication, the administrative cost does not appear to be substantial. Minor changes in ads presently are handled routinely, so the burden of keeping track of the rotational system also should be very small. In addition, rotation by brands should not pose a significant burden because each brand produces its own advertisements.

In conclusion, the system described above could provide consumers with additional information about the dangers of smoking at a reasonable cost to the cigarette manufacturers.

F. "Tombstone" Advertising

The use of imagery is one of the most powerful techniques available to cigarette advertisers. As discussed above,[72] persuasive imagery in cigarette advertising may divert attention away from the health warning. Consequently, France limits the use of imagery in cigarette advertising and allows only the pack of cigarettes advertised to be shown: so-called "tombstone advertising."

Staff believes that limiting the use of imagery in cigarette advertising might significantly reduce the deceptive capacity of those ads. However, this remedy would not provide consumers with information about the hazards of smoking,[73] and may raise potential First Amendment questions. In addition, adoption of an effective informational remedy would correct any current deception by omission in cigarette advertising. Therefore, staff recommends that this remedy not be adopted and not be considered further at this time.

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[72] See Ch. IV, Sec. II.

[73] Only an informational remedy can correct consumer misinformation by affirmatively disclosing material facts.

G. Disclosure of Carbon Monoxide Levels

Another remedy considered by the staff is the disclosure in cigarette advertising of the levels of carbon monoxide gas in the smoke of each brand of cigarettes. Carbon monoxide is one of the most harmful ingredients of cigarette smoke. It causes adverse health effects by reducing the blood's ability to carry oxygen to the cells. Carbon monoxide exacerbates the symptoms of heart disease and chronic obstructive lung disease. CO in cigarette smoke is especially harmful to the developing fetus. Many, if not most, of the instances of prenatal death, complications of pregnancy and long term developmental problems that are associated with smoking are thought to result, at least in part, from the effects of CO. Despite the dangers of carbon monoxide, many people are unaware of its presence in cigarette smoke. In the 1980 Roper Study, 53% of the total sample and 56% of smokers did not know that cigarette smoke contains carbon monoxide.[74]

There are two possible methods for better informing consumers about the presence of carbon monoxide in cigarette smoke. First, if a system of rotational warnings is adopted, a warning informing consumers about the existence of and dangers resulting from carbon monoxide in cigarette smoke should be included. One of the disadvantages to replacing the current warning with a single new warning is that it is unlikely that the warning could be drafted to include this information. Second, CO levels could be disclosed in the same manner that "tar" and nicotine levels are now disclosed in cigarette advertising. The disclosure of CO levels for each cigarette brand might be relevant to consumers in choosing between brands, would highlight the fact that cigarette smoke contains carbon monoxide, and might stimulate competition that would encourage development of low CO cigarette advertising.

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[74] 1980 Roper, q. 30: "Cigarette Smoke contains carbon monoxide, which is a dangerous gas." Total: 31% "Don't know if its true," 13% "think it's not true," 10% "Know it's not true." Smokers: 24% "Don't know if it's true," 19% "Think it's not true," 13% "Know it's not true."

The FTC recently tested different brands of cigarettes for levels of CO.[75] Initial published test results may show a high correlation between CO levels and "tar" and nicotine levels, once a correlation analysis is made. On the other hand, even if there is significant correlation between CO levels and "tar" and nicotine levels, the degree of correlation may vary from cigarette to cigarette. In that case, disclosure of CO levels would provide consumers with useful, additional information. Therefore, it is suggested that this issue needs further study before a recommendation can be made.

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[75] F.T.C., *Report of "Tar", Nicotine and Carbon Monoxide of the Smoke of 187 Varieties of Cigarettes*. March, 1981.

III. LEGAL BASIS FOR PROPOSED REMEDIES

A number of options to remedy the problems discussed in this report have been presented in this Chapter. If at any time the Commission decides to take some action, it is necessary to examine the legal implications of each remedial option it is likely to seriously consider.

A. Proposed Remedies are Reasonably Related to the Elimination of the Deceptive Practices

The Commission has broad remedial authority to enforce Section 5 of the Federal Trade Commission Act. In recognition of the agency's special competence in dealing with trade practice problems, Congress delegated primary responsibility for fashioning remedial orders to the Commission[76] and gave it wide latitude in choosing among alternative remedies to redress violations of its Act,[77] as long as its solutions are reasonably related to the removal or prevention of an unlawful practice.[78]

Affirmative disclosures have repeatedly been found reasonably necessary to cure violations stemming from the failure of advertisements to reveal material facts in advertising.[79]

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[76] "The Commission is the expert body to determine what remedy is necessary to eliminate the unfair or deceptive trade practices...." *Jacob Siegel Co. v. F.T.C.*, 327 U.S. 608, 612 (1946).

[77] *FTC v. Ruberoid Co.*, 343 U.S. 470, 473 (1952); *See, e.g., FTC v. Cement Institute*, 333 U.S. 683, 726 (1948).

[78] *E.g., Jacob Siegel Co. v. FTC*, 327 U.S. at 613.

[79] *Warner-Lambert Co. v. FTC*, 562 F.2d 749 (D.C. Cir. 1977), *cert. denied*, 435 U.S. 950 (1978); *J.B. Williams Co. v. FTC*, 381 F.2d 884, 891 (6th Cir. 1967); *Waltham Watch Co. v. FTC*, 318 F.2d 28, 32 (7th cir. 1963), *cert. denied*, 375 U.S. 944 (1963).

Moreover, the Commission has indicated that affirmative disclosures are "especially apppropriate" in cases, such as this one, involving public health and safety.[80] In fact, affirmative disclosures, such as detailed health warnings, have been required in numerous instances in which the Commission has found advertisements deceptive for containing misleading health and safety claims or for failing to reveal health and safety related facts regarding product use.[81]

In this case, staff has made great efforts to ensure that the remedies discussed are "reasonably related" to any deception in cigarette advertising. Staff has reviewed the existing data concerning consumer knowledge of the health hazards of smoking, and has commissioned studies of its own to assess consumer knowledge. Based on this accumulated data, the single warning and rotational warnings discussed were carefully tailored to remedy the important gaps in consumer knowledge of the health effects of smoking. In addition, staff has tested various warning messages and shapes for both effectiveness in communicating information and understandability, see, Ch. V, Sec. II, supra. It has consulted with social scientists and marketing experts in this regard. Finally, an advertising agency has worked closely with staff to insure that the informational remedies discussed in this report are feasible, effective, and can be implemented at minimal cost and burden to the industry.

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[80] *See* *Firestone Tire and Rubber Co.*, 81 F.T.C. 398, 451-452 (1972), *aff'd*, 481 F.2d 246 (6th Cir. 1973), *cert. denied*, 414 U.S. 1112 (1973); *American Home Products Corp.*, 70 F.T.C. 1524, 1605 (1966); *Kirchner v. FTC*, 337 F.2d 751, 753 (9th Cir. 1964); *Moretrench Corp. v. FTC*, 127 F.2d 792, 795 (2d Cir. 1942); 1964 Cigarette Rule at 8354.

[81] *Positive Products Co.*, 33 F.T.C. 1327, 1335, *aff'd sub nom. Aronberg v. F.T.C.*, 132 F.2d 165 (7th Cir. 1942); *American Medicinal Products Inc.*, 32 F.T.C. 1376, *aff'd*, 136 F.2d 426 (9th Cir. 1943); *Warner-Lambert Co. v. F.T.C.*, 562 F.2d 749 (D.C. Cir. 1977) *see also*, the discussion in *Firestone Tire and Rubber Co.*, 81 F.T.C. at 462-74.

In sum, the informational remedies discussed are directly related to the ineffectiveness of the current warning and the consumers' lack of specific knowledge concerning the health hazards of smoking. As such, they are "reasonably related" to the possible deception in current cigarette advertising, and the Commission has the legal authority to require them.[82]

B. Proposed Remedies are Consistent with the First Amendment

Government regulation designed to cure deceptive advertising is consistent with the First Amendment to the Constitution. In fact, warnings and affirmative disclosures such as those proposed by staff further the First Amendment interest in increasing the amount of truthful information available to consumers.

Several recent Supreme Court cases illustrate these principles. In the landmark case, Virginia State Board of Pharmacy v. Virginia Citizens Consumer Council, Inc., 425 U.S. 748 (1976), the Court stated:

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[82] The "reasonable relation to deception" is applicable whether the Commission proceeds via adjudication or rulemaking. See § 18(a)(1)(B) of the Magnuson-Moss Warranty - F.T.C. Improvements Act, 15 U.S.C. § 2031, et. seq.

> ...[W]uch commercial speech is not provably false, or even wholly false, but only deceptive or misleading. We foresee no obstacle to a State's dealing effectively with this problem. The First Amendment, as we construe it today, does not prohibit the State from insuring that the stream of commercial information flow cleanly as well as freely. 425 U.S. at 771-772 (emphasis added).

In a significant footnote, the Court added that:

> ...[it may be] appropriate to require that a commercial message appear in such a form, or include such additional information, warnings, and disclaimers, as are necessary to prevent its being deceptive. Id. at 772, n. 24 (emphasis added).

In Bates v. State Bar of Arizona, 433 U.S. 350 (1977), the Court also made clear that requiring affirmative disclosures to cure advertising deception does not offend the First Amendment. In overturning a prohibition on advertising by attorneys, the Court acknowledged that the state bar "retains the power to correct omissions that have the effect of presenting an inaccurate picture," and emphasized that "the preferred remedy is more disclosure, rather than less."[83] Again, the Court was careful to note that warnings or disclaimers could be required if necessary to assure that the consumer is not misled.[84]

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[83] 433 U.S. at 375.

[84] See also, Warner-Lambert Co. v. F.T.C., 562 F.2d 749 (D.C. Cir. 1977), in which the D.C. Court of Appeals had occasion to measure a Federal Trade Commission corrective advertising order against the First Amendment. The Court approved the Commission (Continued)

Most recently, in Central Hudson Gas and Electric Corporation v. Public Service Commission, 447 U.S. 557 (1980), the Court confirmed that government regulation of potentially deceptive advertising is not prohibited by the First Amendment. The Court wrote:

> The First Amendment's concern for commercial speech is based on the informational function of advertising. [Citation omitted.] Consequently, there can be no constitutional objection to the suppression of commercial messages that do not accurately inform the public about lawful activity. The government may ban forms of communication more likely to deceive the public than inform it.... 447 U.S. at 563.

Thus, the Supreme Court has concluded that misleading commercial speech is not entitled to First Amendment protection. In this case the proposed remedies solely seek to remedy the potential deception in cigarette advertising. None of the proposed remedies in any way restricts the right of the cigarette industry to exercise its First Amendment right to disseminate truthful information about cigarettes.

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[84] (Footnote Continued)

action, as "the Commission is not regulating truthful speech protected by the First Amendment, but is merely requiring certain statements which, if not present in current advertisements, would render those advertisements themselves part of a continuing deception of the public." 562 F.2d at 769.

IV. CONCLUSION

Based upon its review of these remedial options, the staff has tentatively concluded that there are remedial options available that appear to be capable of providing consumers with the material health information necessary to remedy the possible deception in cigarette advertising. Of the options explored, staff believes the following are likely to be most effective: (a) additional funding for expanded educational efforts, such as public service announcements; (b) changing the shape and increasing the size of the current warning; and (c) replacing the current warning with a system of short, specific rotational warnings.[85]

Expanded educational efforts, such as public service announcements broadcast during prime viewing hours, would reach millions of consumers. Changing the size and shape of the warning would improve its noticeability, but would not provide consumers with the additional necessary health information. Replacing the current warning with a more specific single new warning would be an improvement, but having more than a single warning would allow greater information to be available to the public. Rotating the various health warnings would also assist in maintaining their noticeability over an extended period, and would more effectively communicate a substantial amount of specific health information about which millions of consumers are uninformed. To be most effective these changes should involve the warnings both on cigarette packages and in cigarette advertisements. While the adoption of any one of these remedial options by itself will not eliminate the problems discussed in this report, the adoption of these remedial actions as part of an overall educational effort by Congress, the Commission or other relevant organizations appears to offer the most effective way of informing the public about significant health risks of smoking and eliminating any possible deception in cigarette advertising.

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[85] The staff also repeats the recommendation made in past annual reports to Congress that Congress require the levels of "tar" and nicotine to be placed on cigarette packages, as well as in cigarette advertising.

Committee Note: Due to poor photographic quality, Appendices A, B, and E have been placed in the files of the subcommittee.

APPENDIX C

SWEDISH ROTATIONAL WARNINGS

1. **MOTHERS TO BE:**

   Nicotine and carbon monoxide are transmitted through the bloodstream to the fetus which could be injured by these substances.

2. **A SMOKER'S COUGH IN THE MORNINGS:**

   A smoker's cough is a sign of incipient ill health. Your cough will stop if you stop smoking.

3. **CHILDREN OF PARENTS WHO SMOKE:**
   Are more often prone to bronchitis and pneumonia than children whose parents don't smoke.

4. **LUNG CANCER REAPS MORE VICTIMS THAN ROAD ACCIDENTS.**

   Most lung cancer deaths can be attributed to smoking.

5. **EMPHYSEMA IS A DISEASE:**

   Of the lung tissue which can cause breathing difficulties. Mainly affects smokers.

6. **MORE AND MORE PEOPLE ARE SUFFERING:**

   From heart infarct and other vascular diseases. This applies especially to smokers.

7. **SMOKERS GET ULCERS:**

   More often than non-smokers. Ulcers heal faster if you stop smoking.

8. **PAINS IN YOUR LEGS:**

   Can be caused by smoking which reduces the supply of oxygen to the muscles. Your condition could deteriorate if you continue smoking.

9. SMOKING AND AIR POLLUTANTS:

(E.g., asbestos, radon) make dangerous combination which increases the risk of lung cancer.

10. SMOKY AIR - PASSIVE SMOKING:

Can cause discomfort to people with asthma and other allergies and endanger people with cardiac diseases.

11. SMOKING DURING PREGNANCY AND BREAST-FEEDING:

Can injure your child.

12. DO YOU WANT TO BE IN GOOD PHYSICAL CONDITION:

Your physical condition will improve rapidly if you stop smoking.

13. YOUNG PEOPLE:

The earlier you start to smoke the more dangerous it is. Young smokers can be very quickly affected.

14. IT PAYS TO STOP SMOKING:

It has been proved that people who stop smoking run less risk of poor health.

15. WHAT CIGARETTES ARE MOST DANGEROUS:

Those that give most carbon monoxide, tar and nicotine. Compare the informative labelling on different brands.

16. IF YOU CONTINUE SMOKING:

At least try to cut down, avoid strong cigarettes, leave long butts and, above all, try not to inhale.

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APPENDIX D

The Operation of a Rotational Warning System

The described illustrative rotational warning system was developed in consultation with experts at a New York advertising agency to maximize its effectiveness and hold to a minimum the costs and administrative burdens it would impose upon the cigarette industry and its advertising agencies. As described, the rotational warning system would probably not cause substantial administrative problems for the cigarette companies, would probably not require hiring additional administrative personnel, and would probably not add much to the overall cost of each company's advertising budget.

The proposed rotational warning system could require that each cigarette brand be assigned a list of all of the warnings. The lists assigned to different brands could begin with different warnings so that at any one time each of the different warnings would be in the marketplace. Each brand could then be required to include in all of its advertising for, perhaps, the next three months the warning that was at the top of its assigned list. On a quarterly basis the warning carried by each brand on its advertisements could be rotated to the next warning in sequence on its list. By rotating the warnings on a quarterly basis the administrative and financial expense to the cigarette industry should be held to a minimum.[1]

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[1] Keenan & McLaughlin, Cigarette Warning Project; See Ch. V.

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There are a number of advantages to rotating the warnings on a brand by brand, rather than on a company by company, basis. First, because the system requires different brands to begin their rotational sequence with different warnings, all of the warnings could appear in the marketplace at all times. If the warnings were assigned on a company by company basis, only six warnings could appear at any one time and some warnings might not appear for significant periods of time. Second, by assigning different warnings on a brand by brand basis, each of the warnings could receive approximately the same level of exposure from the beginning. If the health warnings were assigned company by company, those warnings assigned to the smaller cigarette companies, which advertise less or which have fewer brands, would receive less exposure over the short run. Third, assigning the warnings by brand substantially could reduce the risk that any one company would be overly identified with a particular warning even over the short run.

Moreover, it appears that assignment of the health warnings by brand rather than by company could hold down additional administrative or financial burdens. Advertisements are developed and prepared separately for each brand, and each cigarette company generally maintains a separate managerial and administrative staff for each brand.

Solely for the purpose of illustration, assume that there would be sixteen separate warnings as in the Swedish system. The following method of implementing the rotational scheme might be used. Each cigarette company could be assigned a master schedule through a random draw (A-F). Each schedule could contain sixteen lists and

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each list would contain all sixteen warnings. Schedule A would begin with a list which starts with the first warning, Schedule B with a list which starts with the fourth warning, Schedule C with the seventh, Schedule D with the tenth, Schedule E with the thirteenth, and Schedule F with the sixteenth. Each company could then assign one list of all of the warnings in sequence to each of its brands. For example, the company with master Schedule A with the first list starting with the first warning, could initially assign the first list to one of its brands, then assign the list starting with the second warning to a second brand and so on until it ran out of brands. If the company had more than sixteen brands, it could simply start over again at the list beginning with warning number one when it reached its seventeenth brand. The advertising for each brand would then begin with the assigned warning at the top of its list and rotate through the other warnings in sequence. Initially, each manufacturer could decide which of its brands will be assigned which list within its master schedule. Thereafter, whenever a new brand is added to the product line its message schedule would follow consecutively after the company's previous last brand.

The warnings could be rotated in all of the advertising for a single brand on a quarterly basis in accordance with the systems described below for each medium:

Print Media (Newspapers, Magazines, Theater Pamphlets, Magazine and Newspaper Inserts). In each of the print media, all of the advertisements of a particular brand could carry the same warning during each quarter. Every three months all of a brand's print advertising could change to the next warning on that brand's message

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sequence list. The date on the cover of each magazine or newspaper will determine what quarter that publication appears in, and, therefore, which message will be displayed in its cigarette advertising. Using the cover date appears to be the most manageable way to administer the rotating scheme in the print media, because that is the method by which advertising space is purchased. For inserts into magazines or newspapers, the cover date on the carrying publication governs.

Sidewalk Posters, Transit Placards. The rotation of the warning messages in these media would be governed by the actual scheduled appearance date of the advertisement. Transit advertising is routinely changed monthly. Thus, in the month that begins a new quarter, the fact that the replacement ads would include the next warning on each brand's message sequence list should not involve a substantial cost or administrative burden.

Mail Circulars, Give-Away Items, Point-of-Purchase. Items of this nature are usually produced all at one time yet have an indefinite promotional life, making it impracticable to rotate the warning message. Staff considered several options to deal with this problem, because it is especially important for the warnings to appear in point-of-purchase material to ensure the warnings' overall effectiveness. If a health warning did not appear in point-of-purchase promotions, consumers would make the decision to buy cigarettes while exposed to positive images of smoking, but without information on smoking's health hazards. Under staff's proposed system, the particular warning printed on items of this nature will be determined by the date the item is ordered. Thus, a promotional item for a brand of cigarettes would display the same warning as was included on the rest of that brand's advertising on the day the item was ordered.[2] Technically, there will be no rotation of warnings within each of these items. Over time, however, as the cigarette companies continue to produce give-away and point-of-purchase promotional material for each of their brands, all of the warnings would be exposed.

Outdoor Billboards. Staff, in conjunction with the Commission's Division of Compliance, believes that making warnings the length of those proposed as samples clearly visible at the distance from which billboards are normally seen by the passing consumer could take up a substantial amount of a billboard's space. Therefore, a rotational warning system for billboards must take into account the limitations of within-ad disclosures in that medium.

The staff considered several options to provide an effective warning on billboards . One option is to waive entirely the warning requirement for billboards. However, one-half of all billboards are devoted to cigarette advertising. A second option is to require the placement of the identical rotational warnings proposed for other media in billboards, but at a size large enough to be easily read by the public. Depending on the size of the lettering and the warning symbol found appropriate for use on billboards, this approach could take up a significant amount of additional space. A third possibility would be to shorten the text of the warnings for use in billboards, so that the increase in size would consume less space on the billboard. For example, a number of the warnings described in this report as illustrative of the type of warnings the Commission might consider could be shortened for use on billboards as follows:

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[2] There may be some promotional or point-of-sale items that are too small to carry a warning (e.g., matchbooks). Public comment is solicited on how to determine the size of the items below which no warning need be affixed.

"DANGER: Smoking Causes Cancer," or

"WARNING: Smoking Causes Heart Attack" or

"PREGNANT WOMEN: Smoking Endangers Your Unborn Child."

The major problem with this option is that it is not clear that the proposed warnings can be shortened sufficiently to be read on a billboard and still maintain their understandability, objectivety and accuracy. A fourth option would be to permit billboards to be placed without a health warning or with only the new health warning logo, and to require the manufacturers to devote a certain number of their billboards solely to the disclosure of the appropriate rotational health warning in an expanded size.[3] This option permits the most

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[3] Advertisers buy billboard space in each geographic area according to their estimates of the total number of people in that geographic area who will see at least one of their billboards on a regular basis. If the fourth option requiring some billboards to be devoted solely to a health warning is adopted, that option could be implemented by requiring that the health warning be carried on a percentage of each cigarette manufacturer's outdoor advertising budget. To ensure that the warning messages were not placed in the least noticeable locations, the Commission could require that the percentage of illuminated and non-illuminated billboards be the same as the rest of the companies' billboards. Since illuminated boards are generally placed in the best locations, this could mean that the warning message boards would receive adequate exposure. The health warnings would then be rotated on a quarterly basis, in conjunction with the rest of each brand's ads. A quarterly rotation should not (Continued)

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effective display of the mandated health warning. One drawback to this option may be the cost to the cigarette advertiser of placing billboards that do not advertise its product. However, the greater effectiveness of the warning coupled with the lessened interference with the advertiser's message could possibly justify this cost. The fact that the fourth option may cost cigarette manufacturers more than the other options is an issue which merits serious continued consideration. If further investigation or public comment indicates that an effective warning on billboards can be obtained using a less costly option, that option might be preferable.

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[3] (Footnote Continued)

cause a substantial administrative burden because billboard advertisements ordinarily are changed frequently.

Comments on H. R. 4957 - - Proposed
"Comprehensive Smoking Prevention Education Act of 1981"

Domingo M. Aviado, M. D.
President, Atmospheric Health Sciences, Inc.
P. O. Box 307, Short Hills, New Jersey 07078
Adjunct Professor of Pharmacology
University of Medicine and Dentistry of New Jersey
Consultant and Former Member of the
Clean Air Scientific Advisory Committee,
Environmental Protection Agency

The proposed bill, H.R. 4957, states that "Congress finds that ... smoking is the major cause of chronic obstructive lung diseases such as emphysema" [Section 2 (2)]. The statement paraphrases one sentence in the introduction of the Chapter on non-neoplastic bronchopulmonary diseases of the 1979 Report of the Surgeon General on Smoking and Health, which reads "cigarette smoking is the most important cause" (1). However, a careful examination of the chapter raises questions about the validity of that statement because of the complexity of the causation of chronic obstructive lung diseases in general and of pulmonary emphysema in particular. Although there are a multiplicity of suspected pathogenic mechanisms and of etiologic factors, the Surgeon General's Report refers only to those relating to cigarette smoking with an incidental discussion of outdoor pollution, indoor pollution, infections and familial or genetic predisposition. The author of this submission intends to discuss his personal research investigations and those of others questioning the conclusion that smoking is either "the major cause" or "the most important cause" of chronic obstructive lung diseases. Briefly, his reasons are as follows:

(a) Animal experiments have failed to reproduce pulmonary emphysema from long-term exposure to cigarette smoke, although this has been successfully done so for sulfur oxides, nitrogen oxides and other inhalants.

(b) Functional and histopathological studies in humans show uncertain and inconsistent effects of cigarette smoking that do not support the causal relationship between cigarette smoking and chronic obstructive lung disease.

(c) Epidemiologic studies suggest the association of chronic obstructive disease with several risk factors, such as levels of outdoor and indoor pollution, alcohol consumption, occurrence of previous infections, familial predisposition and genetic susceptibility.

A. Experimental Animal Studies on Pulmonary Emphysema.

From 1948 to 1977, the author and his colleagues conducted smoking-related research at the University of Pennsylvania School of Medicine where he was a Professor of Pharmacology. The nature of this investigation was guided in part by comments in the 1964 Surgeon General's Report on Smoking and Health which suggested the need for definitive data on the relationship between cigarette smoking and pulmonary emphysema (2). Although eight of the author's publications were mentioned in the 1967, 1968, 1969 and 1971 Surgeon General's Reports, the coverage was brief and out of context (3-6). Over thirty of his articles on cigarette smoking and over a hundred articles published in the 1970's on related subjects were not mentioned.The government writers cannot be criticized entirely, however, because they have been examining only "Smoking and Health" rather than conducting a global review of all suspected risk factors of chronic obstructive lung diseases. Yet this isolated emphasis on one factor has limited the usefulness of Surgeon General Reports as scientific documents.

a. Experimental Pulmonary Emphysema in the Dog. The 1981 report of the Surgeon General (7) states that "there are no published studies that acceptably show in an animal model that the development of emphysema is induced by cigarette smoking. ... One study in which dogs received smoke directly through chronic tracheotomies reported the development of emphysema [Auerbach, Hammond, Kirman, Garfinkel and Stout (1967)]. The lesions were not conclusive and the results have not been confirmed by others."

The initial series of studies conducted by the author and his colleagues dealt with the effects of cigarette smoke in dogs (8-17). These studies failed to show that inhalation of cigarette smoke caused pulmonary emphysema. It is therefore not surprising that there has been no confirmation of a single study reporting the development of experimental emphysema. It should be noted that the single positive study, although questioned in the 1981 Surgeon General's Report, was highlighted in earlier Reports (including 1979) and may have been responsible for the impression that cigarette smoking is an established cause of pulmonary emphysema.

b. Experimental Pulmonary Emphysema in the Rat. In 1967, the author and his collaborators developed an experimental model for producing pulmonary emphysema in rats (18). Unlike other models reported hitherto, this model permitted functional measurements in addition to histopathologic observations signifying pulmonary emphysema. The exposure of animals to cigarette smoke did not cause pulmonary emphysema (19). Other investigators have also used the same model and have confirmed essentially its acceptability for the study of pulmonary emphysema.

c. Experimental Pulmonary Emphysema in the Mouse. In 1974, the author and his collaborators developed a technique for measuring lung function in the mouse that could not be performed previously (20-22). Chronic exposure to cigarette smoke simulating the dosage of smokers did not cause pulmonary changes signifying emphysema. It is a disappointment to the author that the only study comparing high and low nicotine cigarettes on pulmonary function of mice was overlooked in the 1981 Report of the Surgeon General entitled The Changing Cigarette (7). It is possible that the study was ignored because of the unexpected observations that the lungs seem to adapt to repeated exposure of cigarette smoke.

d. Comparative Studies with Air Pollutants. Dogs, rats and mice are useful experimental models for the study of toxicity of air pollutants. Animals exposed to low levels of products of fuel combustion show impaired lung function and pathological lesions characteristic of pulmonary emphysema and bronchitis. The studies are described in the new series of Criteria Documents that have been prepared by the Environmental Protection Agency on ozone and photochemical oxidants (23), nitrogen oxides (24) and sulfur oxides with particulates (25). This author questions the logic of finding cigarette smoke "the major use" of pulmonary emphysema when primary air pollutants have been shown to cause pulmonary emphysema in experimental animals, and with the same models, cigarette smoking has not.

B. Human Studies on Pulmonary Function and Histopathologic Changes in Cigarette Smokers.

The Chapter on chronic obstructive lung diseases in the 1979 Surgeon General's Report (pages 6-7 to 6-52) starts by defining the terms chronic bronchitis and pulmonary emphysema. For each definition, there is a positive statement on structural or pathological alterations such as hypertrophy of mucous secreting apparatus and epithelial metaplasia as well as more classic histopathological evidence of inflammation for bronchitis; and abnormal enlargement of the air spaces distal to the terminal nonrespiratory bronchiole accompanied by destructive changes of the alveolar walls for emphysema.

These definitions are completely ignored in the remainder of the chapter because functional tests, macrophage changes and enzymatic contents are regarded as pathological or adverse signs of chronic bronchitis or pulmonary emphysema. The definition of adverse and nonadversu effects has been vigorously debated in recent years by the National Academy of Sciences (26) and the World Health Organization (27, 28). The National Academy of Sciences defines adverse effects as responses that are irreversible; the reversible effects are regarded as adaptation or defense mechanism of the lung in response to the inhalant. The predominant opinion is to establish irreversibility as a prerequisite to the definition of an adverse effect. In terms of tobacco use, most of the functional effects described in the 1979 Surgeon General's Report are reversible, not adverse in nature, and are indicative of physiological and biochemical adaptation of the lungs.

a. Respiratory symptoms and pulmonary functional changes. The section on the natural history of chronic obstructive lung disease (pages 6-10 to 6-11) ends with the qualifying statement that pathological data are the most specific and sensitive parameters relating to pulmonary emphysema or chronic bronchitis. It is further stated that "the relationship of early respiratory symptoms to subsequent development of lung disease is unclear" and that "longitudinal studies demonstrating that individuals with abnormal tests of small airways function are at greater risk for lung disease are unavailable". Respiratory symptoms and small airway functional tests therefore are not necessarily indicators of adverse effects or pathological processes.

That smokers show abnormal lung function has not been uniformly observed. In addition to a single study mentioned in the Surgeon General's Report, there are five other publications during the late 1970's that were not cited (29-33).

b. Clearance Mechanisms. The discussion of mucociliary transport (page 6-32) is another example of selective citations from the literature showing negative aspects of cigarette smoking, and omission of conflicting results. Articles that show smoking accelerates bronchial clearance in humans have been omitted. The studies of Albert et al on accelerated clearance in donkeys have been cited, but their article on human clearance stimulation in smokers (34) has been omitted, thus giving readers the impression that the phenomenon has only been seen in donkeys.

Camner, who pioneered in clearance studies in man and whose technique has been adapted by many clinical pulmonary physiologists, has been overlooked. The basic concept that has evolved from Camner's work is that adrenergic stimulation increases mucociliary transport (35), an effect to be expected from cigarette smoking. Patients with predominant pulmonary emphysema but without chronic bronchitis are reported to have no impairment of mucociliary transport (36). Therefore, it is unlikely that smoking, by accelerating airway clearance, would contribute to the pathogenesis of emyphysema.

c. Proteolytic lung damage. The hypothesis that cigarette smoke causes a protease-antiprotease imbalance and in turn potentially leads to pulmonary emphysema is based on a group of selected observations. There is no discussion as to why the hypothesis has not been generally accepted. Turino (37), the first researcher to show that proteolytic enzymes influence the pulmonary mechanics *in vivo*, has noted flaws in the hypothesis that emphysema is caused by the imbalance of protease and antiprotease:

> "An inconsistency for the primary role of elastin alteration as a mechanism for inducing pulmonary emphysema has been a failure to demonstrate alterations in elastin content or composition in human emphysema. Morphologically, elastin fibers have appeared disrupted and diminished, yet most data from human lungs (Johnson and Andres 1970; Pierce and Hocott 1960; and Pierce et al 1961) with the exception of that of Briscoe and Loring (1958), have shown no reduction of parenchymal elastin content by gravimetric techniques."

The above comments can be reinforced by the 1976 observations of Kuhn et al (38) that hamsters treated with elastase intratracheally and developing emphysema also show normal elastin content. Even lung fluid samples collected from smokers and nonsmokers showed no difference in proteolytic enzyme content (38a).

C. Epidemiologic Studies.

Increased mortality from pulmonary emphysema and/or chronic bronchitis among smokers compared to nonsmokers is suggested by seven prospective studies summarized in the 1979 Report (page 6-10). However, these studies are far from being consistent and tend to suggest that many risk factors including cigarette smoking need to be considered in studying the etiology of chronic obstructive lung disease.

a. Air Pollution. The role of air pollution as a risk factor in chronic obstructive lung disease has been the subject of several publications not cited in the 1979 Report. Studies in Tucson, Arizona (39) and in Busselton, Australia (40) showed that an urban environment may contribute to the normal increase in closing volume with age and to the incidence of respiratory symptoms. The higher levels of pollution in England than in the United States has been cited as a cause of the lower ventilatory functional measurements among British males (41).

b. Socioeconomic Status. The illustrative table presented in the 1979 Report (page 6-39) is based on a survey by Higgins of residents of Tecumseh, Michigan. The overall prevalence of chronic bronchitis for various occupational groups ranged from 12.3 for professionals and managers to 30.0 for laborers. An uncited 1977 survey by Lebowitz (42) is more accurate because the diagnosis of emphysema and chronic bronchitis was confirmed by medical examination. His most important conclusion is that smokers do not have a higher incidence of chronic obstructive lung diseases than nonsmokers or exsmokers when the adults were grouped according to income and educational backgrounds. Education and income were inversely related to prevalence of obstructive lung diseases even after controlling for sex, age and smoking.

c. Alpha$_1$antitrypsin deficiency. The summary statement in the 1979 Report that individuals with severe deficiency have the onset of symptomatic chronic obstructive lung disease probably "abbreviated by smoking" was based on selected citations. Two publications in Chapter 6 were not accurately quoted. Chan-Yueng et al and Morse et al did not only examine subjects with mild deficiency but also individuals with severe antitrypsin deficiency which did not deteriorate with smoking. Kidokoro et al (43) concluded in their study of subjects with severe deficiency that "variables other than age and cigarette smoking, influence the severity of emphysema."

d. Other genetic and familial factors. The limited discussion in the 1979 Report needs expansion due to increasing evidence of familial aggregation of chronic obstructive lung disease. The studies of familial prevalence of lung disease conducted by Cohen et al clearly indicate that there is a familial factor unrelated to cigarette smoking and antitrypsin deficiency that is involved in chronic obstructive pulmonary disease (44).

Familial aggregation of chronic bronchitis independent of cigarette smoking, sex, respiratory illness history, residence in common household, geographical distribution and antitrypsin variants has also been reported by Tager et al (45) in 1978. There has also been revived interest in the concept of a congenital or developmental etiology of pulmonary emphysema (46). Additional research directed at the basic cause of emphysema may lead to the identification of additional genetic factors that result in developmental abnormalities leading to adult pulmonary emphysema.

e. Infections. A recent report on the Tecumseh study reveals that acute infection may play an independent role in the pathogenesis of chronic respiratory disease (47). For serologic infection rates for three viruses and M. pneumoniae in males, the incidence of bronchitis is higher for nonsmokers (23%) than in smokers (8.8%). Lebowitz and Burrows (48) arrived at a similar conclusion, i.e. acute respiratory illness is a major risk factor in the etiology of chronic obstructive lung disease.

f. Alcohol consumption. Since 1977, there have been several reports that consumption of alcohol is associated with higher incidence of chronic bronchitis (49-51). In the Tucson study, Lebowitz (52) has concluded that alcoholism is an independent risk factor for chronic obstructive lung disease. There are also earlier observations that alcohol feeding in rats causes pulmonary cellular changes, depression of immunologic reaction, and increased susceptibility to infection (53-55).

D. CONCLUDING REMARKS

It is the opinion of the author of this submission that cigarette smoking is not "the major cause" of chronic obstructive lung diseases, such as pulmonary emphysema. This opinion is based on the author's own research studies and his interpretation of literature on the subject. It is clear that additional research is needed to determine which of the different risk factors for chronic obstructive lung disease are important in a causal sense.

Domingo M. Aviado, M.D.

March 5, 1982

PETER L. BERGER
4 Worthington Road
Brookline, Mass. 02146
Tel. (617) 738-5703

I am a sociologist, currently a University Professor at Boston University. The following is a personal statement, which does not necessarily represent the views of any institution with which I am affiliated. (A brief vita is enclosed.)

My attention was first drawn to H.R. 4957 and S. 1929 by an article in the New York Times on January 30, 1982; I subsequently obtained the texts of the two bills. The latter, in my opinion, constitute a further step in a development that has, for some time now, interested me as a sociologist and disturbed me as a citizen; that is, the growing militancy of the anti-smoking movement and its attempts to control or limit smoking activity.

I have no competence with regard to the medical questions at issue. However, I do claim competence with regard to the broader social and cultural context of this development, and it seems to me that it raises some rather fundamental questions about the scope and purposes of democratic governance on which any citizen has the right to claim competence.

What first interested me in this matter was the aggressiveness with which anti-smoking activities carried on their campaign. It annoyed me as a smoker (though perhaps I should say that I smoke cigars and pipes, not cigarettes, which are the major targets of the anti-smoking groups). No one likes to be depicted as a victim of debilitating vice or a walking threat to public health, and the segregation increasingly imposed on smokers in various public places is frequently demeaning. What also intrigued me, though, was why some anti-smokers were so aggressive about this matter. I could not quite believe that they were all that concerned about me (and other practitioners of this custom). After all, a lot of other things that I and others do are also claimed to be bad for health, yet no comparable furor surrounds them -- and I began to suspect that more complicated motives and interests were involved. I expressed these misgivings in the attached article, "Gilgamesh on the Washington Shuttle," published in Worldview magazine in November 1977.

While this matter has not been a major focus of my professional activities (which, in recent years, have centered on the problems of Third World development), I have maintained an interest in the issue of smoking, contemporary culture and public policy. In the summer of 1979, for example, I attended the Fourth World Conference on Smoking and Health, held in Stockholm under the auspices of the World Health Organization (no doubt an appropriate location considering that the Scandinavian countries have the most stringent anti-smoking policies anywhere). I subsequently served as consultant to a study of the anti-smoking movement in Britain and the United States conducted by Professor Aaron Wildavsky of the University of California at Berkeley. In 1981 I gave a presentation at the Conference on Consumer Policy at the Wharton School in Pennsylvania. (This dealt generally with the evaluation of risks in everyday life; my presentation only mentioned smoking in passing, but it placed the smoking issue in a wider context of the quest for risk reduction.) Consequently, I have a good measure of familiarity with the various arguments made in the course of this controversy.

As to the bills under consideration here, I have no doubts whatever about the sincere intentions of the sponsors. What concerns me is the manner in which such legislation, if enacted, could be used by the anti-smoking forces. My concern is that this would be another step in a long-term campaign of stigmatizing and even criminalizing smoking. First, smoking is stigmatized as a disease; then, smokers are physically segregated; finally, smoking would be viewed as abnormal behavior, to be eventually eradicated or suppressed in all but the most private locations (to be engaged in, if at all, by consenting adults in the privacy of their bedrooms). But what concerns me even more is what this development says about the role of government (in this case, the Federal government) in our lives. It seems to me that those who favor or are considering this legislative proposal should give serious thought to two matters -- the general cultural context and the issue of government power.

The General Cultural Context

The campaign against smoking is not an isolated phenomenon; rather, it must be seen in a much wider context of cultural and social developments in this society. Specifically, it is part of a pervasive quest for security on the part of large numbers of Americans (who, incidentally, share this trait with people in other Western societies). There is a mind-set in this that cannot be unfairly described as institutionalized hypochondria: Life is full of hidden dangers and risks, many of them imposed on the innocent citizenry by rapacious forces beyond their control; this is intolerable; citizens must, therefore, organize to combat these evil forces and, wherever possible, enlist government in this battle. The obverse of this vision of an infinitely dangerous environment is a utopia in which dangers and risks are reduced to a minimum if not eliminated altogether.

The combination of these two themes, one hypochondriacal and the other utopian, makes for a curious ambivalence of timidity and aggression. On the one hand, people with this mind-set see themselves constantly surrounded by terrible perils, many of them deliberately caused or exploited by forces perceived as enemies; on the other hand, the same people become increasingly combative, aggressive and single-minded in the pursuit of their own goals.

One can find this constellation of attitudes in a considerable variety of public issues. But, logically enough, health is a favored focus of these concerns. It is also logical (at least in a psychological sense) that individuals greatly concerned about their own health should be attracted to the anti-smoking movement. Now, I am not saying that it is irrational to be concerned with health; of course it's not. But in the mind-set at issue, there is a tendency to deny that human life, by its very nature, is risky, and to assume that a life-style of passivity would eradicate all risks. As Wildavsky put it well, however, the greatest risk is to take no risks at all. Moreover, the ultimate human risk is death, and that, alas, cannot be avoided. Thus, the utopia of a risk-free existence finally denies the root fact of our mortality. All these attitudes are prominent in the anti-smoking movement. Reading the latter's literature, one gets the impression that, if we only stopped smoking, we would live forever. For example, there is the recurring phrase of "unnecessary deaths" (allegedly caused by the "smoking epidemic") -- a very curious and revealing phrase since, in the end, death is the final necessity for all of us.

I do not want to be misunderstood on this point. I'm not saying that everyone who favors public policies directed against smoking is motivated by such considerations. I am saying that these are themes in the ideology of the anti-smoking movement. I'm also saying that these themes are debilitating both to individuals and to society as a whole. Individuals and societies that dwell on the fearful dangers besetting them will tend to avoid risks of any kind, which is not a healthy posture in the face of the challenges of life.

There is another very important point to be made about the general context of this issue: There are significant class differences both in the general themes just described and specifically in the area of smoking. Lower-income people are more likely to smoke more than upper-income people; conversely, the anti-smoking movement (as are other movements concerned with health and risk reduction of all sorts) is overwhelmingly upper-income in its constituency. Putting this in more sociological terms, the anti-smoking campaign is an initiative coming from the upper-middle-class, while its putative beneficiaries are concentrated in the working class. Put in non-sociological terms, this is but another case of evangelism, in which the better-off classes in our society are trying to impose their own lifestyles on those who are less well off.

There is a long history of this, going all the way back to the early 19th century. The most ambitious case, of course, was Prohibition -- and the long Temperance Movement that preceded it. Here too, well-meaning middle-class people were trying to convert and eventually coerce their social "inferiors". In allusion to George Bernard Shaw's famous play, we might call this kind of cultural imperialism the "Major Barbara complex". Needless to say, working-class people resent this sort of meddling, and they resist it. The same tensions between well-meaning yet aggressive missionaries of a "nice" lifestyle on the one hand and those who are supposed to be the beneficiaries of these efforts on the other hand can be found in the contemporary smoking controversy. It also goes without saying that the upper-middle-class missionaries have greater resources at their disposal in this cultural welfare -- not only resources of money and power, but of education that supposedly legitimatizes their claims to "know better". The bottom line of this pretension is always "We know what is best for you and, therefore, we have the right to tell you how you should live." In everyday situations, the response of working-class people to such elitist interventionism tends to be quite forceful (and frequently unprintable).

Cultural themes are typically related to vested interests of one sort or another. The Temperance Movement kept thundering against the vested interests of the liquor merchants, but in time it was quite correct to speak of "Temperance interests". Similarly, the anti-smoking movement keeps attacking the "smoking interests" (which are, of course, identical with the tobacco interests); but by now one may also speak of "anti-smoking interests" -- that is, the vested interests of those who stand to gain (be it in terms of privilege or power) from the campaign against the regulation of, or the litigation involving smoking. Vested interests, inevitably, have different interpretations of available data. How the evidence may look to an absolutely disinterested observer (say, a non-smoking scientist from outer space) is an altogether different question, but one that, I think, has little to do with the political dynamics of the situation here and now. (One may add that very few people indeed have either the time or the competence to go through the by-now massive body of scientific literature on the subject and to form an objective view of their own. It would seem to follow from this obvious fact that most people, on either side of the controversy, will base their respective positions on something other than rigorously considered scientific judgment.)

These general considerations are relevant in that they lead to the following questions for those who favor this legislation: Do they want to further an overall attitude of unrealistic risk-avoidance in the society? Do they want to stigmatize people and lifestyles that deviate from what is considered proper and wholesome in the elite milieus of the society? More specifically, should public policy and law impose upper-middle-class values and lifestyles on the rest of the population? It seems to me that both realism and democratic values incline one toward negative answers to all three questions. In sum: I don't believe that what American society needs right now is a new version of creeping Prohibition.

The Issue of Government Power

Americans are rightly proud of living in a pluralistic society. In such a society, there will always be conflicts between discrepant values and lifestyles, and evangelism of one's own values and lifestyles is protected by the First Amendment. Also, there will always be conflicting vested interests in such a society, and the idea that these can all be resolved into harmony is utopian and undemocratic. The matter becomes very serious, however, when one ideological party seeks to utilize the powers of government in elevating its own values, lifestyles and interests to monopoly status. In view of the vast powers of the Federal government, this is doubly serious when Federal policy and Federal legislation is involved. In the matter under consideration here, there is "an issue behind the issue" -- the issue of government power. In my opinion, that is the most important issue.

The cultural theme of risk reduction does not necessarily lead to an expansion of government power. After all, there are other ways by which individuals may want to protect themselves from the risks they fear. But, in an age where government is increasingly looked upon as the provider and guarantor of all desiderata in human life, it is only logical that government should be expected to protect people from the risks they want to avoid. In the end, this produces a paradox. Since many of the risks individuals fear are actually incurred as a result of their own actions, government is expected to protect people from themselves -- a curious inversion of classical democratic ideals.

Ivan Illich recently pointed out that the modern state is increasingly taking on the characteristics of the church; it becomes *mater et magistra*, "mother and teacher," the source of all nurture and all instruction. The "magisterial" or teaching function of the state is particularly evident in the legislation under consideration here. Surely, since the first Surgeon General's report on the alleged perils of smoking, the American public has been bombarded with negative communication about this practice, including the warning notice printed, under penalty of law, on all cigarette packages. It is now alleged that the message has not sufficiently registered and that it ought to be reinforced. One detects here the note of irritation familiar to all teachers whose pupils fail to learn the proper lessons. Such irritation, I suppose, is quite justified when children refuse to learn arithmetic or grammar. In this case, though, it carries the disturbing implication that the American public actually consists of child-like individuals. But what is the evidence for this? As far as I know, the only evidence is that smoking has not declined to the degree that anti-smoking activities would wish. But is it not plausible to argue that adults, with all the information available to them, make their own decisions -- including decisions as to what risks to take?

There is something very disturbing about the notion that government in a democracy has a "magisterial" function. It becomes even more disturbing when government pushes its "teachings" ever more aggressively as people don't take to it in the opinion of the "teachers". A democracy presupposes that its citizens are responsible adults; therefore, when that same government engages in activities that treat citizens as recalcitrant children, they have a right to be offended. The proposed policy of rotational warnings has precisely this character of infantilization: "Now, Johnny, since you haven't learned your lesson, you will write it on the blackboard one hundred times . . . ." Magistra indeed!

The most troublesome aspect here, however, is the implicit threat of an ever-deeper penetration of governmental influences in the private lives of individuals. No reasonable person will deny that, in a modern society, government must protect individuals from risks imposed by others. That is, government denies me the right to impose risks on unwilling or unaware others. It is quite a different matter for government to protect me from risks incurred by me alone as a consequence of my own actions.

An analogous case may be instructive here. When I drive my car, I obviously risk injuring other people. The government, quite rightly, seeks to limit this risk. Thus government examines and licenses drivers, imposes penalties for dangerous driving, mandates various safety devices in cars, and so on. No reasonable person will consider such regulation of behavior a violation of the individual's rights. It is quite another matter, though, when government sets out to protect me against the possible risks of my own driving. This issue surfaced in the debate over involuntary safety devices in cars. Should government not only insist that cars contain properly functioning devices that protect others (such as brakes, lights, and the like), but also devices that, regardless of whether I want them or not, protect me, the driver (such as safety belts without which I cannot start the car, and the like)? There has been strong resistance to the latter extension of government regulation, both in the Congress and in the American public. There has been strong feeling to the effect that it is my business and my business only, if I do not protect myself against the risk of going through the windshield in a collision.

Now, it is not my intention to take a position, one way or another, on the issue of involuntary driver-safety devices in automobiles. But I do want to comment on a concept that has been introduced into that debate and that is relevant to the present controversy as well -- the concept of "social costs". It is one of those ideas which, at first glance, seems eminently sensible -- until one starts thinking it through to its logical consequences. Here is what the concept refers to: To be sure, if I risk serious injury or death as a driver, it is myself that I injure or kill. Nevertheless, there are costs to society. Society (be it directly through the state or indirectly through rising insurance costs) has to bear the burden of my hospitalization and medical treatment; society (again, through state or private insurance mechanisms) has to assume costs resulting from my unemployment or disability; if I'm permanently disabled or die, society has to concern itself with the welfare of my dependents or survivors; and the economy has to adjust to my diminished or terminated productivity. From this perspective, I risk far more than my own life or limb in an automobile accident; rather; I risk imposing burdens on others, to the point of threatening (in whatever measure) the viability of the welfare machinery of the state, the viability of the private insurance system, and even the economic well-being of the society (the latter a sort of offense against the Gross National Product).

When one puts the concept of "social costs" in these terms, one becomes (or should become) pensive, for there is a totalitarian thrust to this logic. After all, what actions of mine do not entail "social costs" in the aforementioned sense? I think one must answer: Very few indeed. Thus, my lazy and self-indulgent lifestyle endangers my health (with all the alleged risks to society enumerated above); my child-raising patterns (so say various experts) risk making neurotics or juvenile delinquents out of my children; my hobby of tinkering with household machinery endangers the livelihood of plumbers and mechanics, not to mention my gardening tastes which are a standing offense to the aesthetic sensibilities of my neighbors -- and so on. In other words, once the concept of "social costs" is accepted as a rationale for government interference with private individual behavior, it is difficult to see just where such interference would stop.

This point is so important that a further analogy is appropriate. In the current controversy, there is constant mention of the alleged economic costs of smoking, this supposedly justifying anti-smoking efforts by government. The manner in which these alleged costs are calculated strikes me as rather fanciful (the figures depend on all sorts of presuppositions, medical as well as economic, that seem questionable) but let that pass here. Let me instead propose that excessive and improper eating constitutes a health hazard. Let me suggest that there is a "cholesterol epidemic" in America. If so, let us look at the individual aspects of this first, and then at the possible policy implications. I habitually overeat, and I eat the wrong things. Cholesterol builds up in my body. In consequence, I greatly increase the risk of suffering a heart attack or a stroke. And, if the aforementioned logic is accepted, I incur this risk not only for myself but for others as well; indeed, I'm a walking bundle of potential "social costs". And now imagine the government interventions that suggest themselves in this case: Rotational anti-cholesterol warnings on all food products; prohibition of advertising of cholesterol-rich foods; intrusive government propaganda on matters of diet; government regulation of all meals served in public places and prohibition of certain types of cuisine (say, Italian restaurants); perhaps tax incentives for health restaurants and food establishments; mandatory physical exercise for workers in government or government-subsidized enterprises (in the manner of Maoist China); and so on. Absurd? Maybe (I hope so). But not if one takes the concept of "social costs" seriously as a guide to legislation and public policy. What is missing so far is an anti-cholesterol movement, arrayed militantly against the "cholesterol interests" and with enough political clout to put items from the above list on the agenda. Given the cultural climate I tried to describe before, this may only be a matter of time.

I feel optimistic about the good sense of Americans in resisting this sort of totalitarian encroachment. They showed good sense in resisting Prohibition, made its enforcement impractical, and finally forced repeal. But, in the meantime, a great deal of damage was done. For one, there was an enormous expansion of organized crime -- perhaps inevitable in retrospect, if government criminalizes a large area of private behavior. It seems, though, that the lessons of Prohibition have been forgotten. Be this as it may, it is very important to be alert to the totalitarian implications of all comparable intrusions of government into the private behavior of individual citizens. Most Americans, across the entire political spectrum, recognize the need for welfare-state measures in modern society (political disagreements are really not about the welfare state as such any longer, but about its extent, its mechanisms and its costs). But the accumulatio. of risk-reducing demands on government must bring about a quantum leap in the conception of the welfare state -- a leap precisely in the direction of an all-embracing totalitarian understanding of the role of government. If the behavior of individuals is to be politically proscribed or regulated because it is claimed to carry "social costs" in the aggregate, then virtually no area of private life is immune to government intervention.

Again, I would not like to be misunderstood: I am not saying that the legislation under consideration here is totalitarian either in intent or even in its immediate consequences if enacted. But the totalitarian tendencies in modern society are frequently unintended, incremental, "creeping". That is why they must be resisted in their early, seemingly innocuous, stages. Those who favor the measures proposed by this legislation should ask themselves whether they might not recoil from the final outcome. In other words, the road to a coercive society is paved with all sorts of good intentions; each step on the road may look benevolent and sensible, but the destination may be anything *but* benevolent and sensible.

In conclusion, let me suggest that there is a particular irony to this legislation being proposed at this particular time. Over the last few years, a remarkable bipartisan consensus has developed as to the need to reduce and limit government regulation. It is ironic that, in the name of health, it should now be proposed to increase government regulation in an area of individual private activity. In this particular instance, there is an additional irony, alluded to before in connection with Scandinavian anti-smoking legislation. For a long time, the Scandinavian countries (especially Sweden) were held up as models of the welfare state. Of late, these models have shown themselves to be much less impressive than many people used to think (most importantly, this insight has been growing in Scandinavia itself). It seems to me that we have a very real opportunity in America today to demonstrate that a modern welfare state can be different from the "Swedish model" -- more pluralistic, less costly, less intrusive, working with rather than against private initiative and individual rights. The present case is one of many areas of public policy where, in my view, we can do much better than emulating an obsolete model from abroad.

Peter Berger

Peter L. Berger
University Professor (Sociology)
Boston University*

*Institutional affiliation for identification only

Attached: Vita
1977 article

Observation

Gilgamesh on the Washington Shuttle

Peter L. Berger

The antismoking forces are gaining in political muscle all over the country. Laws and municipal ordinances are passed to prohibit smoking in every sort of public place and, where that is not (or not yet) feasible, to segregate smokers in ever-smaller areas. There is pressure to further restrict advertising for cigarettes and to abolish federal subsidies for tobacco growers. But what is most interesting on the level of everyday life is the aggressiveness and the moral fervor with which this campaign is being conducted. There are segments of the American population in which the hostility to smoking and to smokers has taken on the quality of a crusade. And if we have learned anything from the politics of this century, it is to pay nervous attention to any new crusades appearing on the scene.

I freely confess that I'm not a disinterested observer: I smoke. Also I'm given to political paranoia: I can foresee a time in the near future when smoking will be prohibited everywhere except by consenting adults in the privacy of their bedroom (and, to be honest, I don't trust the antismokers to stop short of the bedroom for very long either). Or, alternatively, I can see myself puffing my cigarillos in a fenced-off area near the toilets in my favorite restaurant, or slinking through the streets with a scarlet "S" pinned on my lapel, or having to make biweekly visits to a social worker to have my permit renewed (you see, I refused group therapy). Let these visions not be taken lightly: I have developed a *wissenschaftliche* respect for my paranoias; most of them have turned out to be predictions, at least when it comes to political reality.

Some stipulations, to be sure, are in order. Undoubtedly there are places where nonsmokers have long been annoyed by the proximity of smokers. Undoubtedly there have been thoughtless smokers who have caused discomfort, sometimes even real suffering, to others. No fair-minded person, even one who smokes twenty cigarillos a day, will object to rules that protect those caused discomfort or pain by people blowing smoke at them. But the current campaign goes far beyond the establishment of such rules. More important psychologically, the aggressiveness of the campaign cannot be explained by such rational grievances. Also, of course, there is evidence to the effect that smoking is unhealthy (though, let it be added, the evidence is less conclusive than the antismoking crusaders would have us believe).

But, once again, the thrust and the mood of the campaign make it implausible to seek motives in concern for smokers' health. Not for a moment do I believe that these people want to protect me from emphysema. As a matter of fact I believe they would be quite delighted if I got emphysema—or worse—as just punishment for my wicked habit. It's sort of the way I assess the motives of Jehovah's Witnesses: They stand on the street corner offering me a copy of *Awake!* because it's their duty to do so. But they *know* that come the day of reckoning very few will be saved—and, the way they look at me, I know that they don't really mind my passing by my chance of joining that élite. In the business of salvation it's long been true that "the fewer, the merrier," and one doesn't have to read Jonathan Edwards to know that the well-deserved suffering of the damned is one of the pleasures of heaven.

I'm digressing. Back to *Wissenschaft*: Sex is in; smoking is out. If you don't believe me read the personals columns in the left-wing press (much to be recommended generally as a depth-probe into the consciousness of the New Class; by their libidos thou shalt know them) and count the number of times that nonsmoking is listed as a trait wanted in sought-after sex partners. It's come to this: "Trim college professor, late 30's, seeks liberated female for complementary relationship [that means he's married]. Tuesday and Thursday afternoons, NYC or Connecticut—*nonsmoker*." "Westchester County couple, happily married, seeks young man or woman, early 20's, for mutually satisfactory explorations in new life-styles—*nonsmokers*." "Hispanic male, currently incarcerated, invites correspondence from submissive females, race immaterial—*nonsmokers*...."

Recently (as the result of a lawsuit, I believe) Eastern Airlines substantially decreased the space allowed to smokers on its planes. The new restriction also applies to the New York-Washington shuttle, a conveyance vital to the existence of politicians, lobbyists, agents of the

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Peter L. Berger is Professor of Sociology at Rutgers University and an Associate Editor of *Worldview*. His latest book is *Facing Up to Modernity*.

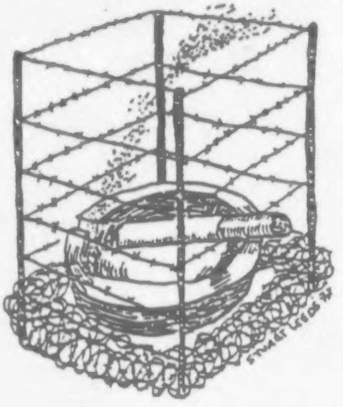
South Korean Government, individuals who carry a hundred thousand dollars in attaché cases, and other people with nervous-making life-styles, mostly males, mostly smokers. But never mind: Justice is blind.

So the other day I was on the shuttle, joining the desperate rush toward the new Jim Crow section in the back of the plane near the toilets, where they keep the oxygen equipment and the old copies of *Business Week*. I lost the race to the last smoker's seat to a fat young man in a seersucker suit, with ten ballpoint pens stuck in his shirt pocket and clutching *two* attaché cases (an agent for both the South Korean *and* the North Korean governments?). Consequently I was forced to sit in rigid abstinence in the enormously enlarged nonsmokers' section (the man next to me, smiling sheepishly, smoked *furtively*—I was tempted to emulate him, but I'm a law-abiding citizen; also I'm given to anxieties, and the stewardess—pardon, the flight attendant—bore a striking resemblance to Bella Abzug...). I suffered (it so happens that I'm also given to fear of flying—Erica Jong hasn't done a thing for me—but that's another story). As everyone knows, suffering is conducive to insight. And so somewhere over Chesapeake Bay (the plane suddenly lurched and the engines didn't sound quite right for a moment there) I had what my teacher Alfred Schutz used to call an "aha experience." I think I know now what is going on in this department of our culture: *Antismoking is the new anti-Semitism. And, like the old anti-Semitism, it has to do with the quest for immortality.*

I recognize that, at first reading, these propositions may lack plausibility. I suggest that the propositions be scrutinized by following a very simple analytic procedure (*Wissenschaft* again): The antismoking attitude is *one item* in the consciousness of a specific group of people; it should be seen in relation to *other items* in that consciousness. At that point, I contend, my propositions gain plausibility. But first: Who are these people? That question is easily answered: We are talking about the aforementioned New Class—the people who consider themselves intellectuals but who are better described as that new segment of the upper middle class that derives its living (and ipso facto its material and ideological interests) from the so-called knowledge industry.

Daniel Bell and Irving Kristol have given us the basic sociological understanding of this class. It is a minority in the American population but, because of its relation to the education and mass communications systems, a very influential one. It is both the matrix and the principal clientele of most of our recent movements of cultural innovation, from the "new consciousness" to the "new politics." Many recent events on the American scene make much better sense if one sees them as part of a class struggle (in quite classical Marxist terms) between this New Class and the old upper middle class (Kristol calls the latter, *tout court*, the business class). The outcome of this class struggle has not yet been decided, but there is little doubt that both the cultural and the political power of the New Class has been on the rise.

Not all elements of the consciousness of this class are relevant to the antismoking issue. But a surprising number are. These are all related to an overwhelming sense of corruption and pollution from which the society is to be cleansed. Ask this simple question: What are *other causes* to which the antismokers are likely to be attracted? There is, probably most important of all, the various causes espoused by the environmentalist movement. It began with Ralph Nader's war against the automobile. It finds its foremost expression today in the campaign against nuclear energy. Modern industrialism (and, in the consciousness of the New Class, especially its capitalist version) is perceived as a gigantic death-machine, destroying the earth, poisoning the atmosphere, spreading every sort of plague. Then there are the causes related to the various health movements.



Commercially produced foods are supposed to poison us, and the only salvation is in the return to some form of organic nutrition. The drugs produced by the pharmaceutical industry (with the possible exception of hallucinogenic ones) are also death-dealing, and the medical establishment has been an accomplice in this conspiracy to destroy us (Ivan Illich—perhaps unintentionally—has helped to legitimate this particular hypochondria). And then there is the strong affinity of this same class for every conceivable type of therapy, from the conventional services of the psychoanalyst to the latest products of the California personal-liberation industry.

Other elements could be mentioned, but these three will do for the present purpose. What do they have in common? Hypochondria is as good a term as any. But one could also put it differently: What is said here is that all of us would lead long and *healthy lives, if only this or that particular source of pollution were removed.*

Thus the cultural scene is rampant with utopias of health: Get rid of the automobile, ban nuclear energy—and we would all live in bucolic idylls, riding our bicycles along crystalline brooks. Eat nothing but food grown on natural manure—and you'll never get ill. Follow this or that therapeutic treatment and you will never be anxious again, never frustrated, never defeated. Is it too fanciful to push these promises to their final limit? I don't think so. For here is the finally implied promise: *Do these things—and you will live forever.* Only if one understands this can one understand the intense hostility to those (be they individuals or "the system") who are believed to stand in the way of this vision of eternal health: *They*—the carmakers, the advocates of nuclear energy, the makers of nonorganic breakfast cereals, those who stop me from having all the orgasms I need—and of course the smokers—*they* are the enemies of life, the purveyors of death, the one great obstacle between me and the redeemed state.

It is not necessary to deny all empirical complaints of these movements in order to make this analysis. Thus, for example, there are very real threats to life in the diffusion of nuclear energy, threats that ought to be taken very seriously in the public debate. In other words, I'm *not* saying that there are no rational arguments on the side of the Clamshell Alliance; I *am* saying that these rational arguments are insufficient to explain the phenomenon of the current antinuclear movement. Indeed, almost every cause has some kernel of truth, but to understand the energizing passions behind a cause the validity of its empirical claims is often beside the point. This is unfortunate, to be sure, but it cannot be helped (those who have not learned this lesson from Marx may learn it from Vilfredo Pareto).

Jean-Paul Sartre, in his book *Anti-Semite and Jew* (incidentally, his last intelligent comment on any politically relevant issue), interpreted modern anti-Semitism as a flight from the human condition. It was a profound and profoundly correct interpretation. For the anti-Semite it is the Jew who is the enemy of life, the corrupting and polluting force eating away at the health of the society. Therefore in battling the Jew the anti-Semite is at the same time struggling for his own redemption, and the victory over the Jew is ipso facto a victory of life over death. This vision, Sartre tells us, is only possible by denying some fundamental and ineradicable elements of the condition of being human. For to be human means to live in an imperfect world, with all the anxieties and ills that go with this. Most important of all, to be human means to be mortal. Thus anti-Semitism, in the final analysis, is a flight from mortality and (in the mode that Sartre called "bad faith") a denial of mortality.

Let me make clear that I'm *not* suggesting a *moral* equivalence between anti-Semitism and these contemporary forms of utopianism. Anti-Semitism, even in its less virulent forms, is a phenomenon sui generis, with a moral perversity all its own that should not be trivialized by throwing it into the same category with a lot of other phenomena. Nevertheless it is very useful to understand the common elements it shares with other turbulences of contemporary consciousness. Nor should we overlook the fact that a new variety of anti-Semitism (still generally camouflaged as "anti-Zionism") has found a response precisely in the utopian milieu being discussed here.

(One significant fact must still be added to the argument: The group that is given to these utopias is almost certainly the most secularized sector of the American population. In other words these are people who have been deprived (or, if you will, who have deprived themselves) of the consolations of religion in the face of mortality and all the other imperfections of the human condition. It is not surprising, then, that the flight from mortality takes on a rather frantic character in this particular ambience. In the absence of God the prospect of death becomes much harder to deal with. There is, of course, the great possibility of stoicism, but very few are capable of that. (Perhaps Sigmund Freud was the last great stoic. Certainly not Sartre, who got hooked on the "bad faith" of revolution.)

And thus it is with us again, that age-old quest for the secret of immortality. Long, long ago Gilgamesh looked for it in his journeys to find the Land of the Living. For a moment he thought he had found the secret in a plant that grew on a faraway shore, and he exclaimed: "This plant is a plant apart, whereby a man may regain his life's breath....Its name shall be 'Man Becomes Young in Old Age.' I myself shall eat it, and thus return to the state of my youth." Then a serpent came out of the water and carried off the plant and (according to the Akkadian version of the Gilgamesh story) "Gilgamesh sat down by the water and wept, his tears running down over his face." One may say that the whole drama of biblical religion is one long effort to wipe away these tears over the anguish of human finitude—but the effort is not an easy one, and its fundamental presupposition is an acceptance of reality and a turning away from the illusions of false promises. It was another serpent, speaking of another plant, who promised: "*You will not die*....When you eat of it your eyes will be opened, and you will be like God."

But let me return from the ancient Near East to the Washington shuttle. We are all sinners. None of us are all that good at coping with the human condition, none of us have sufficient faith. (If I did I'm sure I wouldn't smoke. For what smoking is finally all about is a profound impatience before the mystery of time, and this too is a lack of trust in God who is the Lord of time. But that is yet another story.) All of us are fugitives from mortality. Perhaps we can acquire a measure of tolerance from this understanding: Tolerance of each other—which precludes crusades. And tolerance of the imperfections of the world—which frees us from the illusion of utopianism.

Do you happen to have a light, Ms. Gilgamesh? Thank you. No, I'm not sure either where this plane is going....

STATEMENT

RODGER L. BICK, M.D.

My name is Rodger L. Bick; I am the medical director of the San Joaquin Hematology & Oncology Medical Group. I am also an assistant professor of medicine, specializing in hematology and medical oncology, at the School of Medicine of the UCLA Center for the Health Sciences in Los Angeles. I am on the clinical faculty for hematology and oncology of the Wadsworth V.A. Hospital in Los Angeles, and hold adjunct teaching positions at the Wayne State University Specialized Center for Thrombosis Research in Detroit and at the Wesley Medical Center and University of Kansas Medical School in Wichita.

I received my medical degree from the University of California at Irvine School of Medicine in 1970. In 1973 and 1974, I was the Director of the Hemostasis/Thrombosis Research Laboratory, Chief of Hematology/Medical Oncology, and Director of Medical Education at the Kern County General Hospital in Bakersfield, California. From 1974 till 1977, I worked for the Bay Area Hematology Oncology Medical Group and Bay Area Hematology Oncology Clinical and Research Laboratories in Santa Monica. From 1980 to date, I have been an associate professor of Allied Health Professions and of Nursing and Health Sciences at California State University in Bakersfield.

I am a member of numerous professional societies, including the International Society on Thrombosis and Haemostasis and the American Society of Hematology, and serve as editor for several journals in my field. I have written over 100 published articles on various aspects of the field of hematology.

I have long been interested in the subject of blood coagulation and its possible relationship to heart attacks and cardiopulmonary disease in general. Recent reports have appeared in the literature describing alleged isolation and biological properties of certain brown pigments from cured tobacco leaf, cigarette smoke condensates, and saline extracts of smoke. These pigments have been generally referred to as "tobacco glycoprotein" (TGP). Becker and co-workers have reported that TGP produces allergic skin reactions, induces the formation of antibodies, and activates early clotting factors. Based upon these rather simple experiments, with respect to early blood clotting factors, Becker and co-workers have drawn far reaching conclusions that TGP therefore must also activate the remainder of the clotting system, clot lysing system, and complement system. However, there is no evidence for this sequence of events in either the Becker articles or in subsequent work. In spite of this lack of evidence, these authors have hypothesized that these possible sequential pathological events represent the pathophysiological link between cigarette smoking and cardiopulmonary disease which has been reported.

To investigate more carefully these claims by Becker et al., our group, in collaboration with Dr. Stedman, extracted TGP by the previous published methods of Becker and co-workers. Our group found that the final product obtained by the isolation procedure of Becker contains and consists primarily of a contaminant that is introduced by the methods of separation and appears to be a side product of the photo-polymerization reaction used to form the gels for separation. (Relatives of this contaminant, polymethylmethacrylates, are widely used in orthopedic and dental surgery and traces of these compounds have been implicated in allergic reactions and clot formation.) When we subjected the contaminant to the aforementioned coagulation, clot lysing, complement, and kinin generation assay systems, we found that the contaminant complexed with small amounts of an early coagulation Factor, Factor XII, giving the superficial and completely erroneous impression that activation was occurring. However, it was found to have no activity on thrombogenic assays currently used by the Bureau of Biologics for detecting activation of the blood clotting system. Nor was there any activation of numerous of the early coagulation Factors tested. Likewise, there was no activation of the fibrinolytic system, no effect on the complement system, and no effect on the kinin generating system. Further, we found that there was no effect of this contaminant on the normal clot inhibiting proteins found in human blood.

Our studies strongly suggest that TGP isolated by the general published procedure of Becker and co-workers consists of a contaminant which is introduced during the separation process and contributes to, or more likely, is entirely responsible for the biological activites noted by Becker and co-workers.

The presence or absence of a substance in tobacco smoke with biological properties supposedly effecting the coagulation, clot lysing, complement, or kinin system remains controversial and certainly unproven. It is obviously of importance to examine the significance, if any, of tobacco glycoprotein; however, a good deal of controversy does exist and it has been impossible to duplicate previous findings. In this one instance it appears that erroneous and superficial observations have led to far reaching hypotheses which cannot be confirmed by carefully performed studies. This author's plea would be for adherence to confirmed scientific fact, rather than emotion, in attempting to study and delineate the biological effects, if any, of cigarette smoking on the blood coagulation complement or kinin systems.

My own research and review of the scientific literature lead me to the conclusion that whether and how smoking might cause heart attacks or cardiopulmonary disease remain open questions.

DR. WALTER M. BOOKER & ASSOCIATES
MEDICAL CONSULTANT SERVICES
1725 K STREET, N.W.
SUITE 1401
WASHINGTON, D.C. 20006
TELEPHONE (202) 296-6890

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STATEMENT OF WALTER M. BOOKER, PH.D.

My name is Walter M. Booker. I am President of Walter M. Booker and Associates, Inc., an incorporated biomedical group in Washington, D.C. I am also Professor Emeritus of Pharmacology at Howard University where I was Chairman of the Department of Pharmacology for 20 years.

My post graduate professional training consists of a Master's Degree in Cellular Physiology and Biochemistry from the University of Iowa and a Ph.D. in Physiology and Pharmacology from the University of Chicago. I also spent a postgraduate year as a Senior Fulbright Scholar in Belgium and Sweden, studying with two Nobel Laureates.

I hold membership in numerous scientific societies including the American Society of Pharmacology and Experimental Therapeutics, the American Physiology Society, the American College of Clinical Pharmacology, and the American College of Cardiology of which I am a Fellow.

The views I am expressing in this statement area based upon my training and experience as a scientific researcher in the fields of pharmacology and physiology. In these areas my research endeavors included studies on the effects and mechanisms of action of nicotine on the heart and the autonomic (sympathetic) nervous system.

My main concern with the Bills under consideration, HR 4957 and S 1929, is that they propose congressional action based on findings that have not been clearly established. It is important to note that both Bills make such summary statements as: "Cigarette smoking is the number one cause of lung cancer" and one-third of heart disease deaths "are attributable to smoking." While such opinions are undoubtedly based on some published information, there is by no means unanimity in the scientific community that cigarette smoking can be incontrovertibly labeled as causal.

Despite what those in the legislative arena might believe, the cause or causes of cancer of the lung (and other organs) remain unknown. Both smokers and nonsmokers contract cancer and other diseases often associated with cigarette smoking, and we still don't know why.

The most accurate and appropriate statement that can be made is that cigarette smoking has been identified as one of the many risks or associated factors that may cause or aggravate certain commonly occurring diseases. We are still not able to say whether all relevant risk factors and associations have been identified and consequently, it is practically impossible to control for their effects in experimental studies. While attempts have been made to evaluate certain factors individually, research has not reached the point of being able to say a particular factor is causative.

The scientific fact of the matter is that no one knows why someone like an industrial worker develops cancer of the lung. Is it because he or she is a smoker or because he or she is an industrial worker? Should cigarette smoking be used as a "scapegoat" or should meaningful efforts be made to afford protection to the industrial worker?

Having devoted a large part of my research career to functional underlying processes that might contribute to certain diseases of the heart, I am particularly concerned with those parts of the Bills that seem to ask Congress to decide what specifically causes coronary artery disease. In all sincerity, I raise the question: If smoking has been scientifically shown to be a cause of heart disease, what are the established mechanisms? As a research scientist, I believe that such mechanistic data are necessary before conclusions on causation are possible.

Diet, whether deficiencies or excesses, lack of exercise, emotional stress, genetic factors, auto-immune factors (particularly where cancer is concerned) must each bear its responsibility as a possible cause of disease; but not one of these can be given sufficient responsibility to justify congressional action of the type contained in the Bills being discussed here today.

In summary, I strongly disagree with the proposed Bills because they misrepresent the present state of scientific knowledge. The Bills are asking Congress to give the Nation a scientific verdict on the causes of complex diseases when much of the evidence is either conflicting or has yet to be presented.

STATEMENT OF BARBARA B. BROWN, Ph.D.

I am Barbara B. Brown, former Chief of Experiential Physiology, Veterans Administration Hospital, Sepulveda, California. I received my Ph.D. in Pharmacology from the University of Cincinnati College of Medicine in 1950. I have over 30 years research experience, primarily in the fields of pharmacology, psychopharmacology, neurophysiology and psychophysiology. I began my research activities as a technician in the pharmaceutical firm of The Wm. S. Merrill Co. (subsidiary of Merrill-Richardson) and later became Head of the Department of Pharmacology. After leaving Merrill, I served as Research Neuropharmacologist at Riker Laboratories in California and as Consulting Neurophysiologist at Psychopharmacology Research Laboratories. I was also an associate clinical professor of pharmacology at the University of California Center for Health Sciences in Los Angeles and at the Department of Psychiatry and Human Behavior at the University of California -- Irvine, and also lecturer at the Department of Psychiatry at UCLA. As Chief of Experiential Physiology Research at the VA Hospital in Sepulveda, I was one of the pioneers in the development of the concept of biofeedback. In the past few years, much of my professional activity has centered around attempting to analyze the role of mental activity (brain information processing systems) in health and illness. To date, I have published four books and several theoretical articles on this subject.

Given my extensive background in pharmacology, physiology, and behavior, I disagree with the conclusions and the basic generalizations of House bill 5653. This bill misinterprets the meaning of the reported statistical associations between cigarette smoking and certain diseases. It fails to understand the psychology of the multiple influences that determine individual lifestyles and the acceptance of their risks, and it completely ignores a fundamental and unresolved question: why do people smoke? Further, the bill misapprehends the level of public awareness of the widely publicized "health hazards of smoking," and it relies on the incorrect premise that revised warning labels could substantially affect that public awareness.

Smoking Behavior

Most important is the failure of the bill to address the many unknowns in smoking behavior. A great many perceptive people have observed that smoking behavior is a complex behavior determined by the interaction of several influences, namely, constitutional predispositions, environmental factors, experiential (psychosocial) influences, individual psychophysiologic reaction tendencies, and chemical factors. No single factor determines smoking behavior, nor do all interacting factors influence smoking behavior all the time.

The most convincing study ever conducted on the origins of smoking behavior was the decades-long study of constitutional predispositions to smoking by Seltzer. Using tests to develop psychological and physiological reaction profiles, Seltzer was able to predict who among pre-teens and young teenagers would be smokers when adults. His predictions were about 85% accurate.

Since a major objective of science is to predict events accurately, and Seltzer's work is an outstanding example of achieving this objective, it is unfortunate that the implications of his studies have not been actively pursued.

My own research did not come from considering Seltzer's work, but rather from a continuing interest in the electrical activity of the brain.

A report of my research study on smokers and human EEG activity appeared in the December 1968 issue of Neuropsychologia, an international journal on neurology. The study compared a variety of EEG measures, along with behavioral characteristics, among light, moderate, and heavy smokers, former smokers and nonsmokers. Each subject was studied one full day, under various control and test conditions, for EEG patterns and reactivity.

Results of the study were both unexpected and dramatic. I discovered striking differences between the brain wave patterns and brain electrical responses to sensory stimulation of heavy smokers as compared to those of nonsmokers. Results for the average smokers and for the former smokers were intermediate between the other two groups.

The outstanding characteristics of smokers' EEG patterns are a high frequency, rhythmic, high amplitude, sustained activity. It looks like alpha activity but is twice as fast. Alpha is commonly referred to as the brain wave correlate of relaxed wakefulness and is always contrasted with the nonrhythmic, low amplitude, very fast EEG activity that is commonly correlated with alert behavior. After comparing smokers' patterns with those of former smokers, with those of individuals in various emotional or physical states such as fatigue, etc., I decided that the best explanation for this distinctive EEG frequency is that it represents an inherent tendency for people who smoke to have a level of "alertness" higher than the average and also apparently excess brain electrical activity.

These absolute physiological findings can be interpreted in either of two ways: one, that the high frequency, rhythmic EEG activity is associated with information processing that is concerned with specific elements or thoughts, or that it is helter-skelter, poorly productive, non-linear mental activity. In either case, the smoker consistently shows an excess of (for lack of a better term) "cerebral" energy. "Cerebral" energy can be either productive or nonproductive, i.e., satisfy a predetermined objective or not.

I studied a small group of heavy smokers to learn whether their brain waves would show any effects from short periods of abstinence or any effects when they resumed smoking after abstinence. The heavy smokers who habitually smoked 2.5 to 5 packs of cigarettes per day were asked to refrain from smoking at least 12 hours prior to beginning the experimental recording session. We then intentionally prolonged the recording period in an attempt to increase the "need" to smoke, presumably making the EEG recording experience more stressful. After this, these heavy smoker subjects were allowed to smoke to satiation, and were allowed to smoke ad lib. throughout the remainder of the recording (about 2 more hours).

The results of this sub-experiment were quite surprising. First, no differences in brain wave patterns were found after the subjects had smoked to satiation as compared to patterns found after 12 hours of abstinence from smoking. Second, no changes in brain wave patterns were found at those times when the subjects were actually smoking cigarettes. And third, when the subjects were finally told that they could smoke after the abstinence period, only 1 of the 9 expressed any particular desire or need to smoke. One interpretation of this is that the novelty of the experimental situation acted as an effective substitute for smoking for this brief period of time.

The sustained and marked differences in brain wave activity between smokers and nonsmokers are very probably unrelated to the pharmacologic aspects of smoking. When nicotine is administered to animals, or when the effect of smoking is measured, a stimulant effect is only briefly noted. If the continuing action of nicotine and smoking, especially in "smoking" doses is a tranquillizing one, then the fast EEG patterns of the habitual smokers cannot very well be a result of their smoking activity. The remaining conclusion, then, is that there is a constitutional difference between people who tend to smoke and those who do not.

My studies, and those of Seltzer, comprise powerful evidence that constitutional factors can predispose to smoking behavior or some equivalent behavior that effectively diffuses the excess "cerebral" energy (such as the "high" that runners or joggers enjoy). This does not mean that shaping factors such as genes, home environment, individual experiences, etc., predispose specifically to smoking behavior, but that these factors predispose to a behavior that can discharge excess "cerebral" energy and at the same time be socially acceptable. In other words, people likely to smoke are people who have this particular kind of excess energy -- and smoking provides on acceptable outlet by which that energy can be discharged.

Smoking and Health Studies

The bill's "findings" on smoking and health reflect the government position which is promulgated in the Surgeon Generals' Reports. These Reports are based on highly selective reviews of that literature. The behavioral section of the 1979 Report, for example, admitted this very scientifically uncharacteristic approach. Many excellent "dissenting" reports are ignored. Frequently, when differing reports are cited, they are either inadequately or inaccurately reported. One wonders if occasionally the Reports' authors might not have been biased because of the government's policy on smoking. Unbiased reviews -- and unbiased research -- cannot be fostered in such an atmosphere.

Many research studies cited in the Reports contain serious errors, such as questionable criteria for selecting subjects, poor experimental design, weak criteria for evaluation of results, conclusions not warranted by the data, use of error-ridden or inconsistent public records for information on death and disease, and the abuse of statistics.

One of the weaknesses of these studies that most concerns me is the failure of the epidemiological studies to account for biological variation. In the behavioral area, this translates to the fact that the reasons for smoking differ in different kinds of people. The sum of psychological, constitutional, experiential, motivational factors is never the same for all smokers. There simply is no one, single condition of smoking behavior (incidentally, no single factor accounts for the high rate of recidivism in most stop-smoking programs). Moreover, studies that classify the one pack a day dilettante smoker (one who doesn't care whether he smokes or not, but does so for social reasons) along with the pack a day smoker who sincerely enjoys his smoking but limits it to a pack a day are extremely bad studies from the standpoint of subject selection.

Congress must remember that science is not infallible. Both scientists and scientific reports have normal distribution curves of quality. Very few are excellent; most are just average.

Awareness

In regard to awareness, I strongly disagree with the implication in House bill #5653 that the American public is still too ignorant of the hazards that have been associated with cigarette smoking. I can think of no health claims in the past 20 years that have received more intensive and pervasive media coverage and governmental attention. The media regularly cover all research on smoking and health, particularly reports that suggest hazardous effects. This often amounts to a media blitz.

Further, official warnings are constantly reinforced by anti-smoking commercials and advertisements on radio, magazines, and TV. Advertising by stop-smoking institutes, courses, clinics, etc., constitutes additional media coverage and anti-smoking campaigning. Finally "educational" programs are a fairly permanent part of many school programs. A recent TV news report showed first graders signing oaths that they would never smoke and orally repeating the oath virtually every day. Such a strong effort, which is probably representative of many educational programs, goes beyond merely an education purpose and approaches behavior or thought control.

Warning Labels

Even if I agreed with the bill's findings and supported the belief that public awareness of health hazards was inadequate, I would still disagree with the proposed change in health warnings. As a society, we are extremely uninformed about the basis for behavior that we have termed risk-taking. It should be obvious that the extraordinary sums of money spent on behavioral research, its almost complete failure to discover the causes of these behaviors, and its near failure to prevent such behaviors carry a message. One of the outstanding flaws is the failure of scientists or interested people to encourage studies of personality factors (not neuroticism scores which relate more to neuroses than normal behavior), studies on the role of the environment, studies on the role of psychosocial influences (again the normal rather than the abnormal ones), studies on the role of the human intellect on behavior, and the role of everyday stress on smoking behavior.

Congress must understand that even the so-called experts just don't know enough about why people behave the way they do in a variety of areas, and how that behavior relates to the individuals' health. We need for example tests built on the characteristics of normal people; we must study the normal range of behavior, rather than the abnormal. The majority of smoking behavior studies have used conditioned learning theory. Conditioned learning is an automated, primitive kind of behavior (obviously, since most of the studies are done in rats and pigeons). This theory is unable to explain a behavior as complex as smoking.

Smoking habits have changed, but only a minute part of that change is due to anti-smoking campaigns. People likely to smoke may be people who have excess "cerebral" energy, and our society in recent years has provided them with a variety of other acceptable outlets for that energy. Before any governmental program to reduce cigarette smoking should be considered, a total reevaluation and restructuring of research into smoking motivation and behavior should be commenced. Because of my research, I feel strongly that the reasons people choose to smoke have constitutional origins and this must be taken into account when the advisability of a government cessation campaign is discussed.

Conclusion

House bill 5653 should not become law, because its scientific underpinnings are weak and unconvincing.

Statement

Smoking and Fetal Growth

by Oliver Gilbert Brooke, M.D. FRCP
Department of Child Health
St. George's Hospital
London SW 17

I am a graduate in Medicine of London University, attaining my basic medical degree in 1966. I received an MD degree (British medical equivalent of PhD) from London University in 1974. I am a pediatrician by training, with specialist interest in newborn medicine and nutrition. My present position is Reader in Pediatrics at St. George's Hospital (equivalent of full Professor in the United States) and I am head of Neonatology. Among other positions I have held, I have served as Scientific Officer of the Medical Research Counsel, Tropical Metabolism Research Unit at the University of the West Indies (1969-1972); Member of the Scientific Staff of the Division of Human Physiology, National Institute for Medical Research, Hampstead (1972-1973); Paediatric Registrar and Senior Registrar in Paediatrics at St. Mary's Hospital, London (1973-1976). I am a member of the European Advisory Committee on Feeding of Low Birth-weight Infants which was organized by the European Society of Paediatric Gastroenterology and Nutrition. In addition, I am a member of the following scientific societies: Nutrition Society, Physiological Society, Neonatal Society, British Paediatric Association, European Society of Paediatric Gastroenterology and Nutrition, and Paediatric Research Society. I have been an invited speaker and have presented numerous papers at scientific meetings, and I am the author of numerous scientific publications.

My interest in smoking and fetal growth dates from the early 1970s when I carried out a study on ethnicity and birth size. This involved careful matching of pregnant women for factors such as social status and income, combined with accurate assessment of gestation. Such information is difficult to obtain in large scale epidemiological surveys but is very important in assessing the outcome of pregnancy. When we analyzed our results we found that, after controlling for maternal age, height, gestation of pregnancy, parity, socio-economic status and race, and infant sex, smoking appeared to have little or no influence on birth weight and other measures of fetal growth. (Alvear J & Brooke OG, Archives of Disease in Childhood 53, 27, 1978; Brooke O.G., Lancet 1, 1156, 1977). Since this unexpected finding was at variance with the prevailing views I became interested in carrying out further research in the subject.

A review of the literature of the effects of smoking in pregnancy is contained in the U.S. Public Health Service report "Smoking and Health" (1979, 79-50066). This report marshals a large body of evidence to link smoking with low birth weight. However there is a strong minority body of opinion which holds that the effects observed in humans are mainly mediated through social status or nutrition. The hypotheses here are as follows:

1. Women of low socio-economic status have been known for many years to have smaller babies than more privileged women. Smoking is a class linked practice. There are now far more smoking women in lower than in upper social class groups in Western societies. The link between smoking and low birth weight may therefore not be causal but mediated through other social factors.

2. Smokers may be more likely than non-smokers to have small infants because of their personality or their genetic predisposition to respond unfavorably to stressful events.

3. The effect of smoking could be mediated through nutrition; it is possible that smokers eat less or worse than non-smokers, and that this is the cause of the association between smoking and reduced birth size.

Evidence in favour of one or another of these hypotheses has been obtained by a number of workers over the past 15-20 years:

Yerushalmy produced evidence that smoking was an "index to a particular type of reproductive outcome" (Yerushalmy J, Am. J. Obstet. Gynecol. 73, 808, 1957; Yerushalmy J, Am J. Epidemiol. 93 443, 1971).

Silverman's research supported these conclusions (Silverman D.T. Am. J. Epidemiol. 105, 513, 1977).

Davies et al, in a substantial and well documented study, concluded that much of the effect of smoking on fetal growth was mediated through poor maternal weight gain in pregnancy and was likely to have been of nutritional origin (Davies D P et al, Lancet 1, 385, 1976).

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Rush reported similar findings to Davies in an equally well-conducted study (Rush D, J. Obstet. Gynecol. Br. Cmmwlth. 81, 746, 1974). Rush also found that an effect of smoking on birth weight was confined to women of lower social class (Rush D., Am J. Dis Children 129, 430, 1975). More recently these findings have been confirmed by Papoz et al, who found no effect of smoking on birth weight in Parisian women of upper social class (Papoz L. et al, in: Maternal Nutrition in Pregnancy, Ed. Dobbing J. London: Academic Press, 1981).

I have been involved in research during the last two years to try to clarify these issues. This research has been supported by the American tobacco industry. It involves a team approach (epidemiological, obstetric, pediatric, nutritional and psychological) to the investigation of pregnancy outcome in a hospital providing maternity services to a typical urban community with widely varying socio-economic conditions. The initial 18 month period has been involved with a pilot study of an unselected group of 160 women, examining the feasability of employing various study instruments to evaluate social status, psychological health and nutrition. The results are in the process of analysis.

The next 2 years will, it is hoped, be devoted to answering questions about the relationships between smoking and food intake and between psycho-social stress and birth size. This will lead directly to a large scale epidemiological survey of pregnancy outcome with very careful attention to data collection techniques, and multivariate analysis of factors important in birth size and pregnancy outcome. The techniques used, particularly in relation to the collection of social and nutritional data, will have been thoroughly developed and tested in the initial studies.

Statement of Dr. Victor Buhler

My name is Victor Buhler and I am a pathologist living in Kansas City, Missouri. I am certified by the American Board of Pathology in both pathologic anatomy and clinical pathology. I have held faculty appointments at the University of Kansas School of Medicine and the University of Missouri-Kansas City School of Medicine. I have served as the President of the College of American Pathologists, the Missouri State Medical Association, the Missouri Society of Pathologists and the Kansas City Society of Pathologists. I am a Fellow of the American Society of Clinical Pathologists, the American College of Physicians, the American College of Pathologists, and a member of the International Academy of Pathology and the American Association for the Advancement of Science. I participated upon invitation by President Johnson in the White House Conference on Health in 1965.

At the present time, I am associated with a group of pathologists serving one major hospital and several community hospitals in the Kansas City area, with primary responsibility for the Liberty Hospital, Liberty, Missouri.

I have become familiar over the years with many of the articles cited in the various Surgeon General's reports on smoking and health and with the general medical literature pertaining to this subject. I testified before the Committee on Interstate and Foreign Commerce in 1969 in regard to proposed legislation concerning cigarette labeling and advertising. At that time, I told the Committee that my knowledge of the medical literature, my own investigations and my laboratory experience established "my firm conviction that neither cigarette smoking nor any other etiological agent has been shown to be the cause of cancer of the lung." I have seen no research reports in the intervening 13 years which would change that view. I am pleased to have the opportunity to give you my views on the present legislative proposals embodied in S. 1929 and H.R. 4957.

I am most disturbed by the finding included in both of these legislative proposals that cigarette smoking is the major cause of lung cancer. Such a statement is in my view, not supportable by the pathological and clinical observations I have made during my practice, nor by my review of the medical literature. I am disturbed to see such findings in proposed federal legislation because I believe it is deceptive and misleading to the public and the medical community. The pressure to find causes and cures of cancer and other chronic diseases is considerable, and it should be. The problem, however, is that this pressure creates great temptation to seize upon easy answers. It is always important to realize that objectivity is vitally necessary for scientific progress. The cause or causes of lung cancer are unknown, and a Congressional finding to the contrary does not alter that situation.

Our only hope for progress in the fight against this dread disease is in innovative medical research, not legislative pronouncements.

My view that cigarette smoking has not been proven to be a cause of lung cancer is based on my personal knowledge of the sources for the epidemiological evidence frequently used to support this theory. Epidemiological studies, for the most part, are based on mortality data derived from statements of physicians or others who sign death certificates. In approximately 80 percent of deaths, no post-mortem examinations are done to verify the information on the death certificate regarding the primary or underlying causes of death. Even when autopsies are performed, the results often are not available until after the death certificates have been completed. The recorded causes of death, then, are primarily based on the clinical diagnoses, and it has been my experience as a practicing pathologist that these are frequently incorrect. If death certificates reflected the results of completed autopsies, the statistics would have a much higher degree of validity. At the present time it must be understood that 80 percent or more of all death certificates cannot be considered as having been scientifically validated. Consequently, I have grave doubts about the statistical associations which have been derived from data which is subject to serious flaws.

In addition, the data in statistical studies of lung cancer do not distinguish between primary lung cancer and secondary lung cancer. Primary lung cancer is a malignant tumor originating in the lung. Secondary lung cancer, often called metastatic, is cancer that has spread to the lung from another body site. I have all too frequently found in my own work that tumors clinically considered as primary in the lung are often metastatic and result from cancerous growths in other parts of the body.

One of the reasons why primary lung cancer may be frequently diagnosed when it is not present is that physicians depend primarily on chest x-rays as the technique for diagnosing it. In this regard, it should be noted that chest x-rays present fewer problems in interpretation than x-rays of any other organ or part of the body except the skeletal system. This is because the air in the lungs provides an excellent contrast medium so that disease states are more readily detected. We find, therefore, that it is easier to observe on x-ray an abnormality in the chest rather than, for example, the liver. Even then, however, one cannot always tell from an x-ray whether an area of density in the lung is a nonmalignant or malignant growth, and if malignant, whether or not it originated in the lung.

There are of course, techniques other than x-rays for diagnosing lung cancer. Biopsy and sophisticated scanning techniques (CAT scan), two other routinely used methods, do not always allow for distinguishing between secondary and primary tumors. It should be stressed that primary lung cancer is simulated by secondary tumors arising from other organs of the body with sufficient frequency to challenge the diagnostic accuracy of many deaths certified as primary lung cancer without autopsy exclusion of other primary sites.

In my judgement, the failure of epidemiological studies to distinguish between primary and secondary lung cancer in their statistical analyses raises serious questions about causal interpretations of statistical associations.

Some advocates of the smoking causation hypothesis have said that lung tissue from smokers exhibits certain abnormalities that they describe as "pre-cancerous." They conclude then that cigarette smoking causes these changes which eventually will lead to lung cancer. Let me say first of all that based on my examination of thousands of lungs in microscopic detail that no one can determine whether or not a lesion described as "pre-cancerous" will progress into cancer.

Metaplasia (changes of one type of tissue to another type), hyperplasia (increases in cell numbers), and dysplasia (atypical cytologic changes in cells) can be seen in lungs of both smokers and non-smokers. All of these kinds of changes are rather common and should not be considered as pre-cancerous. In fact, the weight of scientific evidence is that these lesions will not progress to cancer. Metaplasia, common in older individuals, occurs frequently in the trachea or windpipe, and yet tracheal cancer is extremely rare. Any study of autopsy cases that suggests that metaplasia, hyperplasia, or dysplasia provides evidence that smoking leads to lung cancer oversteps scientific bounds. Through my own experience, I cannot confirm that significant cell or tissue changes in the lung occur more frequently in smokers than in non-smokers. Certainly there are smokers without changes, and non-smokers with changes.

As a pathologist, I find the arguments on "pre-cancerous" lesions to indict smoking as a cause of lung cancer to be unconvincing and indeed inconsistent with my own clinical observations.

I might add that, contrary to the reports that one often hears, it is impossible for the pathologist to determine from both gross and microscopic examination of lung tissue whether the person is a smoker or a non-smoker.

I am equally unconvinced by the evidence provided by animal experimentation. As the Congressional Record shows, I twice described to Congressman Tim Lee Carter the critical weaknesses in one of the few inhalation experiments that have supposedly produced cancer -- the Auerbach beagles study. That experiment suffered from severe design defects, and the photomicrographs published with the article would not permit most pathologists to reach the conclusions stated by the authors. I described to Congressman Carter the essential failure of tobacco smoke inhalation experiments to induce lung cancer in animals. That comment is still valid today: no inhalation studies have shown that tobacco smoke inhalation causes lung cancer. Other experimental methods of exposure, such as skin painting, fail to realistically approximate actual human exposure, so that the results of these experimental models must be viewed with suspicion.

Another important aspect of the smoking and lung cancer question involves the histological (cell type) classification of tumors. Some have suggested that the newer cigarettes, generally those with lower tar and nicotine yields, are responsible for the emergence of different frequencies of the various cell types. Squamous cell carcinoma is the type of cancer that has generally been associated with cigarette smoking. It is less frequently found in non-smokers and in women, for reasons that are not yet known. Recently, a few reports have suggested that the relative frequency of squamous cell cancer is decreasing, while that of adenocarcinoma, the type formerly found more commonly in women and non-smokers, may be increasing. I find the interpretation that these new patterns are caused by recent changes in cigarette manufacture to be highly questionable.

First, I believe that the few isolated reports lack sufficient information about such rudimentary factors as nutritional status, occupational exposure, or ethnic background. Consequently, they do not provide, in my view, a reliable measure of the changes in the general population.

Further, of a more technical nature, but of cardinal importance to the pathologist, is the cellular variation produced by the different methods of specimen procurement, fixation, storage, and staining techniques. In addition, the reported frequency of certain cell types will depend upon the source of the specimens; sputum cytology, for example, yields very different results from specimens taken at surgery, which in turn differ from those taken at autopsy.

Most important of all, however, is the fact that there seems to be virtually no solid evidence to link cigarettes with these reported changes. Until more rigorous scientific standards are applied, this change in cell type frequency, if it is real, remains unexplainable and of unknown significance.

Based on my years of experience as a pathologist and on my reviews of the smoking and health literature, I must disagree strongly with the proposed Congressional finding that cigarette smoking is the major cause of lung cancer.

Victor B. Buhler, M.D.

STATEMENT OF

JACK MATTHEWS FARRIS, M.D.

My name is Jack Matthews Farris. I am Emeritus Professor of Surgery at the University of California at San Diego. I practiced surgery in the Los Angeles area for more than 25 years as Staff Surgeon at the Good Samaritan Hospital, Los Angeles; Chief of Surgical Service at the Harbor General Hospital, Torrance, California; and Chief of Surgical Services at the California Hospital Medical Center, Los Angeles. I also served as Senior Consultant to Surgery for the Veterans Administration, for whom I directed the surgical activities at several of their institutions in California.

I am on the Board of Directors and Board of Trustees of the Scripps Clinic and Research Foundation, and was on the Board of Directors for the Medical Research Association of California. I am a member of numerous medical societies where I have served on advisory committees. I have published approximately 73 papers in scientific journals and have contributed several chapters for surgical textbooks. My full Curriculum Vitae and list of publications are submitted with this statement.

Throughout the past 25 years, I have followed with interest the evidence for and against the thesis that cigarette smoking has a causal relationship with cancer of the lung. This interest has been stimulated by my operation on more than 8,000 patients, many of whom suffered from cancer in various parts of the body, including lung cancer.

In 1965, I submitted statements to the House of Representatives and the Senate in which I said that I did not believe that lung cancer had been shown to be caused by cigarette smoking, and that any conclusion to the contrary was not justified.

My opinion today remains firmly the same. There are numerous complexities in the behavior and cause of cancer in all parts of the body. I believe that, when we learn how and what causes cancer, we might well find that cigarette smoking has little or nothing to do with the genesis of carcinoma of the lung.

That the smoking and health controversy continues is demontrated by many studies and findings that cannot be explained by a smoking causation hypothesis. I would like to mention just a few of these.

1. Persons who have never smoked get cancer of the lung that is indistinguishable from those cancers that are reported in smokers. A recent study has shown a significant and increasing incidence of cancer of the lung in non-smokers.

2. In the early 1960's researchers, without regard to smoking in the population, predicted that the death rate in lung cancer would level off in the next decade or so. Studies since then have supported their prediction.

3. Cancer is rarest in those parts of the body most in contact with cigarette smoke, for example, lip, tongue, trachea and larynx. Tracheal cancer continues to be an extremely rare disease, even though the trachea, which is part of the respiratory tract, is more exposed to tobacco smoke than the lung. The larynx is also more exposed to tobacco smoke than the lung; nevertheless, the incidence of laryngeal cancer has remained relatively stable during the last quarter century and has not followed the reported increase in lung cancer.

4. Lung cancer often originates in areas of the lung that have little or no contact with tobacco smoke, that is, in the peripheral areas of the lung, rather than in the hilar, or central, region.

5. Although there is no reason to suppose that there is greater exposure of tobacco smoke at the site of any given cancer in the lung, lung cancer usually appears as a solitary lesion. Metastatic cancer, on the other hand, such as that which spreads from the breast, may produce hundreds or thousands of minute lesions in the lung field. Moreover, once a lobe containing cancer of the lung has been removed, a second primary cancer of the lung rarely develops, although the lung fields have been exposed to the same amount of inhaled smoke.

6. The vast majority of heavy smokers don't get lung cancer. One theory that is being investigated is that the immune system has a lot to do with the development of this disease.

7. Efforts have failed to produce the kind of lung cancer reported to be statistically associated with smoking in tests on animals exposed to smoke. One study has shown that a group of hamsters exposed to cigarette smoke lived longer than those not exposed.

8. Some studies find that in workers exposed to certain chemicals the smokers have a lower incidence of cancer than the non-smokers. For example, this has occurred in workers exposed to chloromethyl ethers.

It is clear that the smoking and health controversy has not been resolved. It is also clear that cigarette smoke cannot be the total answer to cancer of the lung.

All of us who are interested in this controversial subject, have one thing in common - we are all anxious to see a discovery for the prevention of cancer, which inexplicably claims so many lives today. The problem won't be solved by legislating the cause of disease. The problem can only be solved by continued exploration of the various theories and factors suggested by the research to date.

STATEMENT OF SHERWIN J. FEINHANDLER, Ph.D.

I am Dr. Sherwin J. Feinhandler, President of Social Systems Analysts, Inc., a social and behavioral research and consulting firm, in Watertown Massachusetts. I am a consultant and have run projects for federal, state and local government agencies as well as educational institutions and organizations in the private sector. By training I am a Cultural Anthropologist with degrees from the departments of Sociology at Northwestern University, Anthropology at Syracuse University and Social Relations at Harvard University. I have been Assistant Professor in the Department of Psychiatry at Boston University School of Medicine and Assistant Professor in the Department of Anthropology at Boston University. More recently I have been a Lecturer in Anthropology in the Department of Psychiatry at Harvard Medical School.

My research activities have included a focus on social and cultural processes in everyday life, communities and organizations. I have published articles and presented papers on various topics related to these areas. Among my interests is the role of and reactions to tobacco in society. Therefore, I was quite concerned when I heard of proposed House Bill 4957.

The following statement is based upon data collected over several years from smokers, ex-smokers, nonsmokers and anti-smokers on behavior and meaning systems related to tobacco and its use. The data were generated through:

- intensive interviews with people representing all ages, both sexes, and a variety of ethnic, educational and social class backgrounds;
- intensive and extensive group discussions with the same representative segments of the population;
- structured observations of behavior of smokers and nonsmokers in everyday life;
- and recent interviews focusing on decisions made by smokers in relation to their understandings of health claims and smoking.

In our earlier studies we found that smokers and nonsmokers alike were aware of and generally accepted the claims concerning detrimental consequences of smoking. We learned that smoking occurs as customary patterned behavior and has powerful support in both personal and social needs which cannot be readily abridged without serious consequences. Our purpose in conducting new interviews was to determine whether smokers make decisions about smoking based on their awareness of smoking related health claims and warnings. If so, what is the process and what are their reasons for deciding whether or not to continue smoking. In the course of this investigation several issues emerged in relation to these questions. These will be described below.

AWARENESS OF HEALTH CLAIMS

The Surgeon General's reports and the related coverage of their findings have been extremely effective in communicating messages about health and smoking to the public. Our respondents made it obvious that they were keenly aware of the warnings about smoking, usually without our raising the question. When asked her views about the relationship of smoking to health, one respondent's first words were: "The Surgeon General says; 'and whatever he says', smoking is bad for you, and I know it".

Our studies show no tendency on the part of smokers to reject the idea that smoking can be hazardous: "I am convinced it is a problem". For some it is a highly salient one: "I think about it all the time". Even when the issues are less salient for smokers, their concerns are deeply felt. Clearly, smokers know the claims about their smoking, and some attribute a degree of certainty to its consequences which may not be in technical agreement with the facts: "I think (smoking) gives you cancer. What else can I say about it".

Smokers perceive the consequences of smoking to be real, and often imminent even though they themselves feel no negative effects. "Smoking doesn't seem to hurt me. Maybe it is because I have not smoked long enough (to notice any effects)".

Responses to Warning Labels

In the face of this general awareness, our respondents treat health warnings on cigarette packs and in ads as self-evident. When asked about awareness of the Surgeon General's warning on every cigarette package, a respondent commented "All we smokers know smoking is bad". In many instances the warnings are found to be somewhat insulting to the intelligence and raise issues of the motivation behind these attempts to inform. As a consequence, the credibility of the health issues is questioned.

When confronting a situation involving risk, most people engage in a kind of risk assessment in which they attempt to weigh jointly the seriousness of the consequences and the probability of their occurrence. The question of credibility is especially relevant here, since it is raised by some smokers in relation to specific health claims.

Specific statements often raise credibility issues when people have contrary evidence; they then lower their assessment of the probability of risk. They are aware of the specific health claims but disbelieve them.

When asked how they would respond to a warning concerning the relationship between lung cancer and smoking, typical responses of this type were:

> "I don't feel shortness of breath ... I know of lots of cases where people who never smoked got lung cancer and there are so many smokers who don't get it ..."

> "Changing the warnings -- it would scare but then again I laugh because its just not true. Everyone who smokes doesn't get lung cancer. It is just not enough to give up. Maybe I am just stubborn but I don't like being told what to do. The thing that would make me give it up is my own feelings, what it does to my own body."

A more common response among smokers involves some kind of acceptance or belief in the health claims. It is these smokers who engage in a more complex process of deciding whether or not to smoke.

INFORMED DECISIONS TO CONTINUE SMOKING

The fact that smokers continue to smoke, although aware of the claims concerning the health consequences of smoking, may lead to the conclusion that they are engaged in an irrational process. This process has often been dismissed as mere habit. Quite the contrary, we have found that smokers engage in a conscious decision process, not only about whether to continue smoking, but also about whether or not to smoke certain cigarettes through the day.

We have been struck by the general similarity of the process used by people from quite different backgrounds when health considerations are the stimuli for reassessing the decision to smoke. We have described above the approach used by people who question the risk of smoking. Others consider its risks and benefits; we have noted two different approaches. One involves the balancing of two sets of risks: that of smoking against that of not smoking. The other weighs the perceived benefits of smoking against the perceived risks of smoking.

The first approach involves weighing one or more of the predicted consequences of not smoking against the predicted consequences of smoking. The smoker assesses the probability of the consequences on both sides of the choice and jointly determines benefit in terms of choosing which risks are preferred.

> "I tried to give up smoking 4 or 5 times and went maybe three months and put on like 20 to 25 pounds ... for my own health I went back to smoking."

> "I've tried not smoking and found these days to be laden with anxiety. With that choice, I prefer smoking."

The second approach defines the benefits of smoking in terms of the value added to smoking by the uses to which it is put in achieving certain desired ends.

The Sources of Value

We have observed the use of tobacco in fulfilling a wide variety of personal and social needs. Smoking has value for people because of this role.

Lighting a cigarette in response to pleasant or enjoyable experiences, such as the company of friends or at the end of a good meal, is cited frequently. In this instance smoking is said to heighten the experience and enhance good feelings. The pleasure and relaxation many associate with the use of tobacco often derives from the sensorimotor aspect of smoking: taste, handling, visualization of smoke and inhaling and exhaling. One respondent explained:

> "If I really desire a cigarette nothing can replace it. Its not the nicotine. Its the feeling of the smoke going up and down that I enjoy. It is a unique experience. A pleasurable feeling."

People feel that smoking serves to reduce the emotional consequences of negative situations -- when experiencing feelings of fear, anxiety or distress. One informant characterized cigarettes as "a constant companion I carry for comfort."

Smoking plays an important part in the everyday ordering of individual lives. Smoking after a meal is a common practice, witnessed everyday in American society. "I normally stop eating, light a cigarette and then I know that mealtime is over. If I don't have a cigarette, then I feel compelled to eat at least one more thing." Here the act of smoking serves to mark the end of an event. As a structuring mechanism, the act of smoking brings with it a sense of predictability and familiarity which allows individuals to achieve a sense of security.

Smoking serves to impose structure on events which appear to have little or no structure of their own. "I smoke when I have to wait. Its something that helps me get through the waiting."

Cigarettes can aid in the organization of thought. "If I am writing I will stop and have a cigarette to collect my thoughts." Smoking is also viewed as a break from activities, a time in which to relax. "When I work steadily in one place for a long time, smoking is like a little rest period. A five minute break from what I am doing."

Smoking can serve as a social lubricant by providing a common activity for establishing social relationships. In our society the act of offering a cigarette or a light is an expression of a desire to be sociable. Smoking can also serve to affirm social bonds for individuals and groups whose relationships have been previously established.

> "I started smoking in college because there was a real division between smokers and nonsmokers. I was very shy and at certain times after dinner for example people would get together in the smoking room. It was my way to be accepted by these people and to make new friends. And I made a lot of friends through smoking. In the smoking room there was a feeling of cameraderie. You would talk to people that you wouldn't normally. Its a very clear thing, you meet a lot of people through smoking. It was easier to meet smokers than nonsmokers because you had something in common -- the vehicle of smoking."

Smoking is a behavior that a smoker shares with a wide sector of society. The behavior is patterned and the understandings are shared.

VIEWS OF THE PROPER ROLE OF GOVERNMENT

We have illustrated smoking as patterned behavior carried out seperately by individuals over space and time, reflecting tradition and organizing concepts in common. Smoking, deeply ingrained in our culture through centuries of tobacco use, has thus become an accepted custom in society. This explains why the majority of smokers and nonsmokers share similar views about the government's role in relation to smoking.

These views can be summarized in terms of the social value placed on freedom of individual choice and the absence of arbitrary regulation when social processes can work out the issues at hand. There is also a feeling that externally imposed regulation removes responsibility from individuals and natural social groups. While some people rather like the idea of avoiding responsibility, they hesitate in agreeing to the inevitable sacrifice of free choice.

Most respondents feel that the appropriate role for government in relation to smoking is that of keeping people informed.

> "I feel that the government has the responsibility to warn people because no single person can accumulate all knowledge and conduct tests. I feel that government should conduct studies, I just don't feel that the government has the right to make your choice. Telling you you can't smoke is like telling you, 'you can't have an alcoholic beverage'. No attempt to do

> this would work, is silly and wastes a lot of money. All it does is to threaten the credibility of the laws of our country. Like prohibition, it would create a black market. It becomes common practice to break the law. It would make law-breakers out of people that are otherwise considered good citizens. Cigarettes are even less dangerous than alcohol because they don't have the potential to create deviant behavior."

The Attribution of Deviance

Current attempts to regulate the smoking process are seen as part of an effort to define smoking as deviant behavior. Many people recognize that the attribution of deviance is extended to the smoker himself.

> "I began to smoke at 24; there was nothing terrible about it then. There was no publicity. I can hardly enjoy it now. I feel very weak minded."

> "It seems to me that people are now trying to make moral judgements on smokers and the government is backing them. They try to put guilt on the smoker and it is the smoker who suffers."

Noteworthy in this respect are recent efforts to encourage businesses to discriminate against smokers in hiring and the establishment of a psycho-diagnostic category, "smoking behavior disorder".

Anti-smoking groups and government are felt to be active in this effort. Smoker and nonsmoker alike view the efforts of anti-smoking groups as deviant rather than viewing smoking as such. They question the motivation of people in these groups and attribute to them either Messianic or power seeking desires. Government attempts to restrict customary behavior are similarly questioned. People thus feel that government demonstrates hypocrisy, inappropriate exercise of power and undue manipulation of individual rights.

> "I really think that the government and the people who are anti-smokers like to point fingers. It makes them feel good. 'I'm not doing that, therefore I must be better than they are. I must be a better person'. There is no difference between smokers and nonsmokers. To categorize smokers or nonsmokers is ridiculous. We have enough discrimination in the U.S. now. Why try to create more. But it is anti-smokers against the rest of the world."

> "I think that this is all political. I think that there are people out there who are violently anti-smoking, not for health reasons and they have money

> and a lobby. If it was for health reasons they would be on the bandwagon for a hell of a lot of other things. People are bothering me and it is a personal insult. They shouldn't be allowed to bother me. Anti-smokers have no personal consideration or sensitivity. It is a legal way for them to relieve a lot of aggression. I may hate a woman wearing strong perfume but I don't pour water on her or tell her to get off the elevator because she is bothering me. There has to be something more going on. Its looking only after themselves; I am good and they're bad."

Most people reject the attributions of deviance applied to smokers because they recognize that smoking is part of customary behavior and place a high value on free choice in matters of custom. Many nonsmokers, even those who have experienced discomfort as a result of others' smoke in specific situations, have said that interpersonal processes are to be preferred to legislative restrictions.

SUMMARY

Smokers are keenly aware of the publicity surrounding the relationship between health and smoking and the warnings on cigarette packs. Given this awareness, they show no tendency to reject the idea that smoking can be hazardous, whether or not they have any desire to give up smoking. The sheer volume and intensity of the warnings has led many to question the motivation behind them, and the validity of their content. Specific warnings encourage smokers to consider easily found counter-examples.

This kind of behavior is often dismissed as irrational, however, it is a highly logical response to social needs and realities. Smoking is an indispensable aid in encouraging positive social behavior, and enabling individuals to deal with situations in the world about them.

The fact that people continue to smoke in the face of seemingly overwhelming publicity against smoking is proof, in itself, of its social importance. It follows that attempts, which smokers perceive to be unjustifiable coercion and hypocritical at best, involves social consequences whose costs are incalculable.

S.J. Feinhandler, Ph.D.

STATEMENT

OF

H. RUSSELL FISHER, M.D.

I am H. Russell Fisher, of Glendale, California, a doctor of medicine. I am Emeritus Professor of Pathology at the University of Southern California, and former Director of the Department of Pathology at the Memorial Hospital of Glendale — where I am now on staff as a consultant. Until last year, I was Director of the Department of Pathology at the Santa Fe Hospital in Los Angeles, where I am still on the active staff as a research associate. I am a member and past officer of medical and scientific societies, including the National Research Council Committee on the International Council of Societies of Pathology. My special investigative interest has been in the field of carcinoma *in situ*. My full Curriculum Vitae is submitted with this statement.

As a medical scientist, I am concerned with the legislative proposal now termed the "Comprehensive Smoking Prevention Act of 1982." It would be inappropriate to adopt it on several grounds. Its basis is contrary to a significant body of scientific knowledge about the putative role of cigarette smoking in the causation of disease. It misuses the scientific definition of "cause" as that term relates to disease. Its justification rests in part upon statements that have not been found to be valid. Moreover, the bill creates an unnecessary additional layer of bureaucracy, duplicating many functions already in place and available in various agencies in the government; this may well hamper solid scientific research. The National Institutes of Health and the National Cancer Institute are already expert in reviewing grant applications.

In 1969, I submitted a statement to Congress in which I said that I, like many of my fellow scientists, was puzzled over the cause of lung cancer, and that, on the basis of the evidence then available, no one knew the cause of cancer of the lung. Contrary to the proposed "findings" in the bill, we still do not know the cause or causes of lung cancer; nor do we know what role, if any, smoking plays in the causation of this disease.

Most of the claims made against cigarettes are based on statistical coincidence, or statistical association, from which conclusions can be drawn only if all the facts of nature are known, which they are not. This statistical coincidence has given rise to an emotional tide, seized upon by various groups and agencies, in the hope to fill a void in our efforts to achieve a perfect health.

Like most pathologists, I am not expert in the use of mathematical systems or statistics, but there are a few basic rules that are easy to apply. For example, each population group used in the statistical analysis must have uniformity and homogeneity. Data must be reliable and comparable and, most importantly, the reference assumptions must have an existence in fact.

Most, if not all, of the epidemiological studies reporting a statistical correlation between cigarette smoking and lung cancer do not meet these simple criteria. For example, they rely primarily on death certificates for the diagnosis of lung cancer even though studies have shown great unreliability in the death certificate specification of this disease. This is primarily due to confusion between cancers that arise in various parts of the body and spread to the lungs - metastatic cancer - and cancers that start in the lungs - primary cancer.

Sommers, participating on a committee of the New York Academy of Medicine to evaluate the accuracy of death certificate diagnosis, found them to be correct in less than 50% of the cases. And Rosenblatt, in a series of studies on the diagnosis of patients dying in the Doctors Hospital of New York during the last decade, found that the clinical diagnosis of primary carcinoma of the lung was confirmed by autopsy examination in only 45% of the cases.

In 1979 Saxen reported on the difficulty of achieving accurate death certificate diagnoses, working with data from Finland, a small country with a stable population. Reviewing 162 deaths attributed to respiratory cancer in a 2 year period, he found, on microscopic tissue examination, that 35 cases (22%) had to be ruled out as not having primary lung cancer.

One thing that has added to the difficulty of this problem is the controversy about the various histological types of lung cancer and their relationship to causation. Some years ago Kreyberg, a famous lung pathologist of Oslo, classified cancer of the lung into two major types or groups. One group was primarily made up of squamous cell carcinoma and the other adenocarcinoma. In squamous cell carcinoma the cells grow as flat plates, like the cells of our skin. In adenocarcioma the cells are cylindrical and grow in rings, like glands.

Kreyberg proposed the idea that the reported rise in the incidence of lung cancer was due to a rise in incidence of squamous cell carcinoma and that this kind of lung cancer was caused by smoking and other environmental factors. According to Kreyberg, adenocarcinoma was unrelated to environmental factors. Somehow this idea that smoking caused some types of lung cancers but not others was accepted by many, even though they could not agree with Kreyberg on his standards for classification.

Because many investigators associate cigarette smoking with squamous cell carcinoma, there has been continuing interest in the trends of squamous cell carcinoma versus adenocarcinoma. The study by Vincent et al., from Roswell Park showed that since 1963 squamous cell carcinoma has dropped and adenocarcinoma has increased. In contrast, a Mayo Clinic study showed a progressive increase in squamous cell carcinoma. Such differences are probably due to a lack of uniformity in the standards of microscopic criteria, and statistical selection emphasize the need for homogeniety in elements subjected to statistical comparison.

The controversy about tumor types is particularly pertinent to the issue of lung cancer in women. It is claimed that the incidence of female lung cancer is rising due to their increase in smoking since World War II. However, studies indicate that the purported rise is primarily due to an increase in adenocarcinoma, the type not generally associated with smoking. For example, Dr. Ioachim from Dr. Sommers' pathology department at the Lenox Hill Hospital (N.Y.), finds that adenocarcinoma makes up about half of the cancers in females.

That the smoking and lung cancer controversy continues is amply demonstrated by the recent study which appeared in the April 1979 issue of the Journal of the National Cancer Institute. This study challenged the dogma that smoking is the major cause of lung cancer. Using data from a number of broad studies of lung cancer, including the American Cancer Society study population, Enstrom showed that there has been a dramatic rise in lung cancer mortality rate among persons who have never smoked. In white females who have never smoked, the increase in lung cancer in the last 4 decades has paralleled the increase in those who smoke cigarettes. This negates the claim of the American Cancer Society that the increase in lung cancer in females over that period has been due to cigarette smoking. Enstrom noted that "Many factors other than personal cigarette smoking have not been exmained in great detail in their relationship to lung cancer."

We just do not know the cause of lung cancer despite a mountainous accumulation of research on the subject. Scientists have not produced the kind of lung cancer associated with smoking — squamous cell carcinoma — in animals exposed to cigarette smoke. Experimental work simply does not support the idea that lung cancer is caused by cigarettes and scientists are looking elsewhere for the cause of cancer, into genetic, environmental, and viral possibilities. The federal government, through the National Cancer Institute is now funding research into these possibilities.

There is no need to create a new office of smoking and health to collect the results of research on the effects of smoking, or to facilitate such research. This could be done adequately by capacities already in place in the Department of Health and Human Services by assignment to a single qualified individual qualified in bibliographic compilation and by a directive from the Head to those organizations already expert in reviewing grant applications, such as the National Institute of Health and the National Cancer Institute. A new bureau just isn't needed nor is it necessary or appropriate to have a new complex committee on educating the public until there is a firmer foundation of scientific knowledge of what is to be taught, what reliable information is to be disseminated.

Knowledge from medical and scientific research must be interpreted with great care and with an understanding of the great variability of the biological processes. Any action having the force of the Congress of the United States should be based on fact, not conjecture. Experience has shown that social legislation not based on valid information has an unhappy history.

Statement of Katherine McDermott Herrold, M.D.

My name is Katherine McDermott Herrold. I am a certified pathologist and am presently retired from my most recent position as medical director of the United States Public Health Service.

I received my medical degree from Woman's Medical College of Pennsylvania in Philadelphia in 1948. From 1948 through 1949 I interned at George Washington University Hospital in Washington, D.C. Between 1952 and 1955 I was the chief medical officer at the Federal Reformatory for Women in Alderson, Va. Between 1955 and 1957 I was a resident in clinical pathology, and between 1957 and 1959 I was a resident in pathological anatomy, both of these positions being at the National Institutes of Health in Bethesda. Between 1959 and 1971 I was a laboratory pathologist at the National Cancer Institute in Bethesda, Maryland.

I am a member of numerous professional societies including the American Society of Clinical Pathologists, the College of American Pathologists, the International Academy of Pathology and the American Association for Cancer Research. I am a member of the honorary medical society of Alpha Omega Alpha.

I have published papers in the scientific literature dealing with pathology and cancer research.

Those who assert that cigarette smoking causes lung cancer rely on the data contained in several prospective studies, including one whose data was first presented by Dr. Harold F. Dorn in 1958. That study reported a statistical association between smoking and lung cancer. However, Dr. Dorn recognized the grave danger of relying upon death certificates for lung cancer diagnoses without pathological confirmation. It was my privilege to work with Dr. Dorn on this project in providing the clinical and pathological review of the data.

The Dorn study involved a total of almost 300,000 U.S. veterans who were policyholders of U.S. Government life insurance. I reviewed the clinical and pathological materials available from the 2,241 patients with the diagnosis of lung cancer on their death certificates. Histologic material was available for review in about 65% of those cases. Extremely important from a biological standpoint is the fact that only a small percentage of even heavy cigarette smokers develop lung cancer.

The frequency of the histologic types of cancer was also established. Of the 472 patients who were "current cigarette smokers" at the time of their deaths, and for whom histologic sections were available for my review, I found no correlation between the various histologic types of lung cancer and the amount of tobacco smoked. Further, the age at death from lung cancer was not related to the age at which smoking started, the number of years smoking, or the number of cigarettes smoked per day.

These findings were in agreement with those of R.D. Passey some ten years earlier. Dr. Passey wrote that it could be said without hesitation that the age at death from lung cancer is not that which is associated with chemical carginogenesis. Passey's findings, as well as those of Doll and Hill, were almost identical to mine - that the age of diagnosis of lung cancer is not related to the age at which the patient started to smoke, the length of time smoking, or the amount smoked.

Those who make claims against smoking assert that there is a dose response relationship between smoking and lung cancer; that is, the risk of developing lung cancer increases with the number of cigarettes smoked and the earlier one begins to smoke, the greater the risk. My findings, like those of Dr. Passey before me, suggest otherwise.

In recent years, there have been reports of a so-called "epidemic" of lung cancer, which some have attributed to increased tobacco consumption. However, scientists have noted that this apparent increase may well reflect changes in the detection rate rather than in the actual incidence of the disease. The diagnostic problem is exacerbated by reason of "detection bias" among smokers, i.e., the fact that smokers undergo a more rigorous examination for lung cancer than non-smokers. Studies reporting that lung cancer occurs more frequently in smokers than in non-smokers all assume that the non-smokers are studied with an equal frequency and intensity as the smokers. However, Professor Alvan Feinstein of the Yale Medical School found this not to be the case at various institutions, including the Yale-New Haven Hospital. Hence, he concluded in 1974 that "cigarette smoking may contribute more to the diagnosis of lung cancer than it does to producing the disease itself."

The various anomalies found my myself and others in the population studies combined with the problems associated with diagnosing lung cancer, lead me to the conclusion that more research is needed in this field before we can accept as proven the theory that smoking causes lung cancer.

UNIVERSITY of PENNSYLVANIA
PHILADELPHIA 19104

The Wharton School CC
DEPARTMENT OF STATISTICS

(215) 243-8222

Statement of Richard J. Hickey, Ph.D.

I am Richard J. Hickey, a senior research investigator in the Department of Statistics of The Wharton School, University of Pennsylvania, Philadelphia. I received a Ph.D. degree in biophysical chemistry and microbiology at Iowa State University, Ames, Iowa, in 1941. Thereafter, I performed research and development work on antibiotics, vitamins and other biochemicals. In the mid--1960's, I became involved in research on the air pollution/public health problem at the University of Pennsylvania. Since 1973, I have been investigating the relationships of environmental chemicals and other variables to chronic disease risk at the University's Wharton School. Many of the journal articles and book chapters published, of which I was author or co-author, pertain to environmental pollution.

The following comments concern both H.R. 4957 and S. 1929. A basic criticism of both proposed acts is the implicit assumption that "findings" (1), (2), (3), and (4), which are used to justify both the labeling statements and the proposed actions, are known and proven. However, there are serious scientific questions about the scientific bases for the claims that cigarette smoking causes the various conditions associated statistically with smoking, such as lung cancer, heart disease, and low birth weight.

Documentary scientific evidence establishing causality is not given in the proposed bills. However, it is clear from the wording of the "findings" that they rely heavily upon the alleged causality evidence presented in some publications and reviewed in recent U.S. Surgeon Generals' reports on smoking and health. Unfortunately, these reports do not represent an even-handed review of the available evidence. There are several reasons for rejecting the conclusions of these reports, including:

(A) *Selective Reporting or Selective Exclusion*. Selective reporting is present in the Surgeon Generals' reports. Sources that conform to the pre-determined smoking-causality position of the reports are cited extensively, while studies that challenge or discredit this position are often selectively excluded. Warnings of the misuse of statistics have also been excluded. The Surgeon Generals' reports have the characteristics of briefs for the "prosecution" only; the "defense" has been largely omitted even though these reports should have been unbiased.

(B) *Uses and Misuses of Statistics*. The Surgeon General's report of 1964 asserts (p. 182): "Statistical methods cannot establish proof of a causal relationship in an association. The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability." These two statements lie at the root of the problem. The first sentence is a correct statement; causality cannot be established by statistical associations. This is a standard caveat that is taught in elementary statistics courses. The second statement is false. Subjective judgment or personal opinion cannot be used to interpret the causal implications of a statistical association, no matter how strong and no matter what level of significance. Judgment and opinion do not constitute substantive evidence.

It should be recognized that statistical associations are mathematical quantities. When properly obtained, they may *suggest* possible causal relationships, but they can *never* prove such relationships. Human conditions such as cancer, heart disease, spontaneous abortions, stillbirths and birthweight deficiencies are all basically biological and biochemical problems, not mathematical or statistical problems. When ill, one visits his physician, not his statistician.

Properly used, statistical methods are capable of rejecting an hypothesis to the extent that it is incompatible with observed data, but statistics alone can *never* establish that an hypothesis is certainly true. This fact is widely misunderstood. However, recent Surgeon Generals' reports have rather routinely inferred causality from statistical association, *e.g.*, that cigarette smoking causes lung cancer if the two variables are positively associated in the statistical sense.

Statistical associations may be due to chance or to dual influences of some third factor. For example, it was reported in 1952 that "there is ... a significant correlation in the United States between the population to physician ratio and the population to bed ratio and the number of reported deaths from cancer of the lung." [R.E. Rigdon and H. Kirchoff, "A Consideration of Some of the Theories Relative to the Etiology and Incidence of Lung Cancer," *Texas Rept. Biol. Med.* *10*, 76-91 (1952) (p. 88)]. Surely no one would conclude from this correlation that physicians "cause" lung cancer.

It is known that cigarette smokers tend to be coffee drinkers; there is a positive correlation between measures of cigarette and coffee consumption in some populations. Is it to be concluded, therefore, that smoking "causes" coffee drinking, or that coffee drinking "causes" smoking?

Another fallacy is the presumption by some that non-random samples are not biased. This belief is especially hazardous when working with genetically heterogeneous populations such as of humans. For example, some studies that employ self-selected samples such as those of heavy smokers, light smokers, non-smokers, and ex-smokers are likely to be biased. This point was addressed by T.D. Sterling

["The Statistician vis-a-vis Issues of Public Health," *American Statistician* *17*, 212-216 (1973) (pp. 212-213)]: "The manner of selecting populations to study the effects of smoking has been criticized by some of the world's most respected statisticians but to little avail (Berkson, 1958, 1960; Fisher, 1957, 1958; Mainland, 1956; just to mention a few). Of concern to statisticians has been the major reliance on 'volunteers' in most of these studies and the consequent multiple sources of serious bias. Ordinarily, results from a study such as Doll's in which over 30 per cent of the queried population did not participate would be considered, if at all, with greatest suspicion, especially if, as is freely admitted, the responders were quite different from the reference population. (Doll, 1964). Similarly, the data collected by volunteers of the American Cancer Society and used to link smoking to a variety of diseases stem from a study population (also of volunteer families) that dramatically differs in most significant comparable measures from the U.S. population (Sterling, 1971a, 1972a)."

(C) *The Genetic/Constitutional Alternative*. In the late 1950's, Sir Ronald Fisher, a former President of the Royal Statistical Society, suggested the following alternative to the smoking-causality hypothesis: "Cigarette smoking and lung cancer, though not mutually causative, are both influenced by a common cause, in this case the individual genotype." The problem of resolving the conflict between the smoking-causality hypothesis and the genetic/constitutional hypothesis was recently reviewed in depth by P.R.J. Burch ["Smoking and Lung Cancer: The Problem of Inferring Cause (With Discussion," *Journal of the Royal Statistical Society*, Ser. *A* (General), Vol. *141*, Part 4, pp. 437-477 (1978)]. Burch concluded (p. 476): "The discussion has allowed me to undermine one causal model and it has revealed divergent opinions that might surprise those nurtured on the reports of the Royal College of Physicians and the Surgeon

General of the United States. I am also happy to record that the constitutional theory, proposed by a former President of this Society, the late R.A. Fisher, is alive and well."

To this date the genetic/constitutional hypothesis has not been discredited. It has, however, been denigrated subjectively and widely ignored. Despite the fact that Burch's paper appeared in a major statistical journal, it has not been cited in any Surgeon General's report. Further, the constitutional hypothesis has been "criticized" by some who would seem to equate criticism with rejection. Some criticism has been of the "do not like" variety.

Unless the genetic/constitutional alternative hypothesis is rejected on valid scientific grounds, it would be risky to base public policy upon the smoking-causality hypothesis.

If more anti-smoking legislation is passed and if eventually it is established that such habitual smoking is in fact symptomatic of an underlying genetic trait that both influences smoking behavior and contributes to increased risks of developing those diseases associated statistically with smoking, then the U.S. government would be placed in a totally untenable position. The prospect that R.A. Fisher, considered by some to be the father of modern statistics, has been correct all along is very real and cannot be ignored.

(D) Air Pollution and Chronic Disease. In addition to the omission of publications such as that of Burch, the Surgeon Generals' reports have also extensively excluded from consideration published epidemiological reports on the complex relationships between a number of common urban air pollutant chemicals and mortality rates for several categories of cancer, heart disease, and other causes of death in U.S. cities.

Some reports showed that concentrations of several common air pollutants such as sulfur dioxide, sulfate, nitrogen dioxide, and trace metals in the air of 38 cities were significantly associated statistically with lung cancer mortality rates for these cities. In fact, over 70% of the variance in lung cancer mortality rate was explained statistically by air pollutant characteristics of these cities. Further, over 50% of the variances in mortality rates for several other cancer classifications were also explained statistically by air pollution data. Also, over 50% of the variances in mortality rates for certain heart disease classifications was explained statistically by air pollutant concentration data for these cities. Significant relationships were also found between air pollutant concentration data and mortality rates for infants under one year of age and mortality rates for congenital malformations. [See, for example, R.J. Hickey *et al.*, "Ecological Statistical Studies Concerning Environmental Pollution and Chronic Disease," *IEEE Transactions on Geoscience Electronics*, Vol. *GE-8*, 186-202 (1970); R.J. Hickey, "Air Pollution," pp. 189-212, in: *Environment: Resources, Pollution & Society* (W.W. Murdoch, Ed.), Sinauer Associates, Stamford, Conn., 1971; R. Mendelsohn and G. Orcutt, "An Empirical Analysis of Air Pollution Dose-Response Curves," *Journal of Environmental Economics and Management*, Vol. *6*, 85-106 (1979)].

(E) *Comments on Four "Findings" of H.R. 4957 and S. 1929*. The following comments refer to "findings" (1), (2), (3), and (4) that appear on page 2 of both bills. The greatest detail is offered regarding finding (4) which pertains to pregnancy, prematurity, low birth weight, and related problems of infant health.

Finding (1). H.R. 4957 states that "cigarette smoking is the largest preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually." This statement pertains in large part to deaths from cancer and from cardiovascular diseases. However, it says nothing about the constitutional hypothesis nor about epidemiological and other evidence regarding the relation of air pollution to chronic disease.

Finding (2). S. 1929 states that "smoking is the primary cause of lung cancer" in the United States, and "is associated with other cancers." This claim has been made in various ways by the Surgeon Generals' reports. However, as stated previously, this claim is defective because it involves in part selective reporting, misuse of statistics, ignores evidence regarding the genetic/constitutional influences, and fails to consider published evidence regarding strong statistical relationships between air pollutant chemical concentrations and cancer mortality rates. It is also a fact that, despite many attempts, lung cancer has never been induced in experimental animals in well-designed studies by fresh tobacco smoke when experimental conditions resembled those of natural exposure.

As mentioned above, deficiencies in the theory that smoking causes lung and other cancers were examined at length by Burch in 1978. In 1965, K.A. Brownlee ["A Review of 'Smoking and Health'," *Journal of the American Statistical Association 60*, 722-739 (1965)] examined the 1964 Surgeon General's report, *Smoking and Health*, and found it defective in biochemistry and statistics. It is noteworthy that reports claiming that the smoking-mortality associations are causal appear largely in medical and biomedical journals rather than in statistical journals. In contrast, reports in statistical journals do not accept as valid the inference of causality from statistical association. It is also noteworthy that Brownlee's review was not mentioned in several subsequent Surgeon Generals' reports.

Finding (3). S. 1929 states that "heart disease accounts for nearly one-half of the deaths in the United States and one-third of the deaths attributable to heart disease are associated with smoking." Death rates from heart disease increase rapidly with increasing age, especially beyond 40 years. Thus, heart diseases appear to be in large part diseases of aging, a fact not evident in this finding.

The statement that one-third of these deaths are "attributable" to smoking implies cause. "Associated with" does not. There is no question about the association in some populations, but anyone who then argues for a causal relationship makes a huge leap based on faith alone. Any causal implication is defective because it involves in part those factors discussed earlier. In addition, quite strong statistical relationships between concentrations of a number of common air pollutant chemicals and mortality rates for several classifications of heart disease have been reported. The air pollution - heart disease relationship has often been overlooked or minimized in Surgeon Generals' reports.

Finding (4). S. 1929 states that "the risks of miscarriage, stillbirths, premature births, and child weight deficiencies for pregnant women who smoke are higher than for pregnant women who do not smoke." The term, "risks", is strictly statistical and is not biological or biochemical. The implication is that because of statistical associations, the smoking-causality explanation is true. This is the same misuse of statistics, and of science, noted previously.

Causality cannot be inferred from statistical association. The causal implications or claims, as stated in the Surgeon Generals' reports, are also defective because of selective reporting. Among the published reports that address the problem are: R.J. Hickey, R.C. Clelland, and E.J. Bowers, "Maternal Smoking, Birth Weight, Infant Death, and the Self-Selection Problem," *American Journal of Obstetrics and Gynecology*, Vol. *131*, 805-811 (1978). Misuse of statistics is pointed out along with other errors and fallacies, including the overlooking of or subjective denigration of the publications of the late Professor J. Yerushalmy.

That low birth weight has been associated statistically with maternal smoking during pregnancy in some populations is not in question. However, the implications or claims in the Surgeon Generals' reports that the association means smoking-causality constitute misuse of statistics. Factors other than smoking also correlate with low birth weight, such as certain socio-economic variables, maternal height and weight, geographic differences, and altitude variations. The 2,500 gram birth weight criterion, implying that less than this weight is unhealthy, ignores the large variations in these factors. For example, there is evidence that birth weights of large, tall women tend to be heavier than birth weights of children of small, shorter women. [See J.M. Tanner, L. Lejarraga, and G. Turner, "Within-Family Standards for Birth-Weight," *Lancet* *1*, 193-197 (1972)]. Surely constitutional factors influence birth size and weight in many populations.

An informative but controversial kind of evidence that addresses the smoking--causality versus the constitutional hypothesis pertains in part to whether women who later take up smoking ("future smokers") tended to have low birth weight children even before they started smoking. This question was examined by Professor J. Yerushalmy ["Infants with Low Birth Weight Born Before Their Mothers Started Smoking Cigarettes," *American Journal of Obstetrics and Gynecology*, Vol. *112*, 277-284 (1972)]. He found that, indeed, birth weights of children of future smokers tended to be lower than birth weights of children of never smokers. Such a finding is in accord with the constitutional hypothesis rather than with the smoking-causality hypothesis. Yerushalmy's findings were consistent with those reported by D.T. Silverman [*Maternal Smoking and Birth Weight*, Thesis, Johns Hopkins University, Baltimore, 1972]. These findings have not been widely popular.

It is also noteworthy that Yerushalmy found that the smaller babies of smoking mothers were healthier than the smaller babies of non-smoking women.

To summarize, in view of selective reporting in the Surgeon Generals' reports, the evidence supporting the constitutional hypothesis, the misused statistics and the sometimes biased samples, it seems desirable to table any legislative actions until the Congress has received a more complete analysis of the evidence. At the present time, statements about deleterious effects of smoking are often put forward supported by evidence that is scientifically unconvincing and possibly invalid.

Richard J. Hickey

Richard J. Hickey

STATEMENT
OF
ROBERT CASAD HOCKETT

I am Research Director of The Council for Tobacco Research -- U.S.A., Inc. I have been employed by the council and its predecessor, the Tobacco Industry Research Committee, since 1975, first as Associate Scientific Director, then Acting Scientific Director, and in my present capacity. My Curriculum Vitae is attached.

In my opinion, the proposed amendment to the Federal Cigarette Labeling and Advertising Act will cause justifiable consternation among many earnest and able investigators of cancer, heart diseases and emphysema at the experimental and clinical levels. These scientists labor in the hope of clarifying the etiology and pathogenic steps and stages in the development of these disorders with a view to preventing or delaying their clinical appearance. Contrary to the "findings" in the bill, however, the cause or causes of these diseases have not been scientifically established, and much work remains to be done.

Statistical Association

The claims against smoking are based largely on epidemiologic studies. Positive statistical association, however, -- as epidemiologists are aware -- does not mean causation. Accordingly, these studies do not support the "findings" in the proposed amendment.

Students of logic have long recognized that a negative statistical association between an event and a putative cause may be conclusive. Thus when it was suggested that some hair dyes might produce cancers in women, a study of 5,000 beauticians who had been exposed to such dyes for twenty years showed that these persons had not experienced any excess of cancer. This negative correlation appears quite conclusive with respect to dyes that have long been in use. Similarly a concern regarding the SV-40 virus, which causes bladder cancer in monkeys and was found to be present in some polio vaccines, was considered resolved when no higher cancer rate was found in the vaccinated humans than in the unvaccinated ones.

On the other hand, a positive statistical association between an exposure and a disease condition is well known to be only evidence of some sort of a relationship, the nature a of which remains to be discovered. For example, there is a statistical association of severe overweight in women with higher uterine and ovarian cancer rates and in men, of higher colon-rectum and prostatic cancer. In the same category is the positive correlation between early marriage (or sexual activity) of women and eventual cancer of the cervix. Such positive associations only indicate a need for other kinds of investigation in order to elucidate the nature of the relationship.

Still more striking is the reported epidemiological finding that men and women who average seven hours of sleep per night have lower death rates from coronary heart disease than those getting either more or less sleep. Those getting less than five hours had very high death rates and those sleeping ten hours or more per night had higher than average rates.

I have previously pointed out that merely changing the hours in bed of the short-term or long-term sleepers to seven per night, would be unlikely to change this picture radically. More likely the amount of "useful sleep" that an individual can achieve is a reflection of a psychophysiological diathesis that may be the basis of the difference and which may or may not be alterable. Studies show that many persons are able to achieve "useful and effective sleep" in a relatively short period. Others may have difficulty in getting to sleep or sleeping restfully on account of psychological tensions or emotional problems. Whether such persons can be treated to reduce such tensions is a question many investigators are attempting to attack. Surveys have shown that many smokers testify to the feeling that smoking cigarettes helps to arouse them when they are drowsy but to relax them when they are tense. Confirmation of these contradictory impressions by objective pharmacological methods has been difficult, but studies of electro-encephalograms have claimed correlations between certain types of brain waves and psychological characteristics of the subject. Some subjects can learn to produce various types of brain waves (electro-encephalograms) at will. Others have difficulty in producing the types reputed to reflect inner repose. Smoking has been claimed to promote this kind of tranquility but the evidence so far is widely regarded as "soft". The discovery of centrally active small peptides (endorphins, encephalins and related substances) may eventually provide an answer to these questions.

Lung Cancer Diagnosis

In 1912, a book by Adler called attention to the occurrence of primary carcinoma in the lung and apparently created the impression that this disease was a new development whose cause must be sought. By coincidence this book appeared near the time when development of the blended cigarette in the United States was stimulating a vast expansion of cigarette use in this country, which was further stimulated by the social conditions brought about by World War I. Thus, a basis was laid for the claim promulgated a few years later that an "epidemic" of lung carcinoma had been engendered by cigarette smoking.

Meanwhile, however, the late Dr. Milton B. Rosenblatt had made a very intensive and extensive study of lung cancer as described in the medical literature, mostly European, of the nineteenth century where the art and science of pathology was developed and practiced relatively early. The application of post-mortem examinations for cause of death was made quite routinely in some central European hospitals long before it became prevalent in other areas. Carcinoma of the lung was recognized, post mortem, in a great many cases where it had been missed altogether in clincial pre-mortem diagnoses. In fact, it was missed clinically as often as 90-95% of the time. In hospitals where post mortem histological diagnosis was practiced regularly, carcinoma of the lung constituted a proportion of total male cancers similar to or greater than that reported today. Yet in that era cigarettes were practically unknown.

In retrospect, it is not strange that lung cancer was missed in clinical diagnosis. Tuberculosis was common as a source of bleeding from the lung and death from pneumonia generally occurred in the lung carcinoma patient so that cancer was not suspected.

As better clinical diagnostic methods came into use, such as radiography, bronchoscopy, exfoliative cytology, exploratory thoracotomy and others, the gap between clinical diagnosés and post-mortem discovery of lung cancer was closed gradually over several decades. The introduction of antibiotics eventually played a role also since a pneumonia might be resolved, exposing a carcinoma to discovery.

In the United States, this European work was apparently little known. The dogma was that all lung cancer must be metastatic so that the primary lesion must be found if the origin of the neoplasm was to be known. Moreover, post mortem examination was much less frequently made than in Europe.

Although histological examination of cancer tissue by an experienced pathologist still provides the most reliable diagnosis available, the development of clinical methods that are quicker and easier apparently discouraged the application of painstaking post-mortem study in this country, and has promoted the general use of less reliable clinical methods.

The lung is a frequent target for metastases from other organs. Dr. Shields Warren reported that about one-third of all cancers arising in other regions of the body eventually metastasize to the lung, often at a early stage. Dr. Rosenblatt thought it very unlikely that such metastatic cancer present in the lung but not originated there was "caused" by tobacco smoke. He felt it logical to exclude metastatic cancer in the lung from any statistical study of correlation with smoking. But the distinction between primary and metastatic cancers in the lung is not always easy and a distinction is no longer required on death certificates or attempted in statistical records. While the distinction appears to be very important for the study of causative factors and etiology the necessary data are not easily available.

Nevertheless in a careful review of contemporary death certificates and hospital records in a few individual hospitals, Rosenblatt found considerable clinical over-diagnosis of lung carcinoma in patients who were known to be cigarette smokers. Similar findings have been made by Feinstein.

Animal Experiments

When the first large-scale epidemiological studies of the association between cigarette smoking and lung carcinoma were described publicly in the mid-fifties, a number of investigators undertook to expose many different species of animals, including mice, rats, hamsters and hens, to cigarette smoke inhalation. None of these early inhalation studies produced lung carcinomas. The 1958 Annual Report of the British Empire Cancer Campaign referred to these experiments as adding up to a "striking negative result."

The first wave of smoke inhalation studies was so unproductive of results that other methods were sought. It was recalled that the black tar accumulated as a by-product from destructive distillation of coal had been shown to produce skin cancers when painted on mice and rabbits. Accordingly, researchers began applying tobacco smoke condensate on the backs of mice. Condensate was obtained by puffing cigarettes mechanically and passing the smoke into a cold trap where it was condensed into a dark-colored viscous liquid. Though this is not "tar" as defined in the dictionary and does not bear any close resemblance to coal tar, the term persists.

It must be pointed out that, contrary to popular belief, tobacco smoke condensate -- or "tar" -- is not actually a material to which human smokers are exposed. Many chemical studies have shown that there are qualitative, as well as quantitative differences between laboratory condensate and fresh smoke to which humans are exposed.

Nevertheless, researchers began painting such tobacco smoke condensate on the backs of mice, generally dissolved in some solvent. After persistent treatment over a long period with enormous doses, neoplasms did appear on the skins of these mice. This method of testing was seized upon by numerous other investigators with many variations of technique, different species and strains of animals and varying results. Some of these experiments were not reproducible. Chemists fractionated the tars and by the proliferating techniques of chromatography identified literally thousands of chemical components of cigarette smoke condensate with a high degree of reliability. Probably no other complex mixture in the human environment has ever been so thoroughly analyzed.

I was critical of this development from the start. A "typical" human smoker draws a puff of air through his cigarette. This generates a cloud of smoke that enters his mouth within a fraction of a second following formation, remains in his oral cavity for another fraction of a second and then is drawn into the lung diluted with five to ten volumes of air.

During this short interval, rapid physical and chemical changes are taking place. The tiny liquid droplets that constitute the blue cloud are growing in size by coalescence, which influences deposition in the lung. Numerous compounds in the aerosol are polymerizing, interacting, combining, breaking down, and otherwise changing. If such a mixture enters a cold trap, the most readily condensed substances wil be deposited as a thick liquid but other phases of the smoke escape into the air.

This situation complicates the design of devices for exposing animals to inhalation of smoke comparable in physical and chemical properties to that inhaled by human smokers. The best we can do is to design devices that will produce the smoke mechanically under conditions approximating those attained by humans and get it to the animal's lung at a comparable age and in monitored dosages.

These considerations led me to characterize the skin-painting of mice with cigarette smoke condensate as applying the "wrong material in the wrong form, in the wrong dosage, to the wrong tissue of the wrong animal." I still hold the same view.

To the best of my knowledge, to date no one has produced the type of lung cancer that is associated with human smoking by exposing experimental animals to inhalation of fresh, whole smoke. The considerations, experiences, trials, tests and findings of twenty-eight years that have been described in extreme brevity will, I hope, make it clear why The Council is now emphasizing study of the "constitutional" diseases as such on a basic level with the help of new research tools. I have tried also to show why dogmatic positions are inappropriate and restrictive in the present state of knowledge.

Statement
of
Duncan Hutcheon, M.D., D.Phil.
Departments of Pharmacology and Medicine

My name is Duncan Hutcheon. I am Professor of Pharmacology and Medicine at the College of Medicine and Dentistry of New Jersey. I am also an attending physician at the College Hospital in Newark and am director of the Clinical Pharmacology Training Program at the New Jersey Medical School, where my responsibilities have included serving on the Human Studies Research Committee. I am a graduate of the University of Toronto where I received my M.D. and B.Sc.(Med.) degrees. In 1950, as a National Research Council (Canada) Postdoctoral Fellow, I obtained my Ph.D. at Oxford University in the Department of Pharmacology.

My research has been primarily in the field of cardiovascular and clinical pharmacology, and I have approximately 65 research publications. In addition, I have contributed sections of several textbooks, including Drill's Pharmacology and Medicine, Treatment of Heart Disease in the Adult by Rubin, et al., and Cardiovascular Therapy by Russek. Since 1977, I have served as editor of the Journal of Clinical Pharmacology.

I hold memberships in numerous medical and scientific societies including the American Society for Pharmacology and Experimental Therapeutics and the Society for Experimental Biology and Medicine. I am a fellow of the American College of

Physicians and the American College of Clinical Pharmacology of which I am a past-president.

In May, 1980, I helped establish the Princeton Institute of Environmental Medicine (PIEM), an organization of medical scientists concerned about the health effects of chemical substances in the environment. Our research focuses primarily on the relationship between various environmental risk factors and chronic diseases such as heart disease and cancer. The environmental factors we are investigating include tobacco smoke constituents, industrial chemicals, pesticides, and emissions from jet, diesel, and gasoline engines.

It is the consensus of those associated with the work of the Institute that it is impossible to make strong, unequivocal statements about the contribution of any single factor to the occurrence of cancer and heart disease. For this reason, many of the statements in H.R. 4957 and S. 1929 regarding the health and economic consequences of smoking are unacceptable scientifically and appear to represent an attempt to offer easy solutions to what are actually complex multifaceted problems.

Furthermore, it is our belief that Congress has the responsibility of encouraging thorough and unbiased investigation into the spectrum of risk factors associated with these diseases rather than limiting its consideration to only one aspect. Why, for example, doesn't the proposed legislation mention environmental hazards? After all, in terms of both quantity and inherent carcinogenicity and cardiotoxicity, environmental chemicals such as polycyclic hydrocarbons (PAH's), halogenated hydrocarbon pesticides and industrial solvents appear to pose serious public health problems.

These views are based on our field work that involves correlating mortality data provided by the National Institutes of Health with our own clinical and laboratory findings. By this method, we have examined the following environmental pollutants: benzo(a)pyrene (BaP) and other PAH's, carbon monoxide (CO), and halogenated hydrocarbon pesticides and industrial solvents.

Our studies show that there is considerable misinformation regarding the possible biologic effects of the various chemicals to which we are regularly exposed. Moreover, there is little appreciation of the way in which such substances are handled by the body (i.e., how and when they are eliminated), and the relationship between the amounts of chemicals present in the environment and the amounts necessary to initiate carcinogenic and cardiotoxic activity.

For example, we measured serum B(a)P levels by radioimmunoassay procedure in urban and suburban population groups in the New York metropolitnn area and found significantly higher levels in the group living in the areas with highest atmospheric pollution. B(a)P was chosen as a marker of urban air pollution because it is a suspected carcinogen that is produced by burning fossil fuels. It is also considered to be a valid indicator of potentially carcinogenic PAH's in the atmosphere. Our observations indicate that geographic location may be an important risk factor for respiratory cancer. While these results represent only preliminary findings, they suggest that environmental studies should be conducted on a much larger scale. Such studies could assist in identifying populations at environmental risk and pinpointing geographic areas where environmental control efforts should be directed.

During the past three years, my laboratory has also focused on the effects of environmental chemicals on cardiac performance. This has involved investigating the association between geographic location and the prevalence of heart disease and sudden cardiac death. Our findings suggest that certain chemicals associated with adverse cardiologic effects are present both in the ambient air over Newark and in the drinking water supply of suburban communities. The chemical agents that may be related to cardiac mortality rates in areas of greatest risk are the halogenated hydrocarbons.

Although we originally assumed that CO might play some role in heart disease causation, our findings have suggested that (1) CO does not appear to predispose the heart to catecholamine-induced arrhythmias in experimental animals; and (2) although the CO levels are higher in the ambient air in the urban-industrial section of Newark than in the New York suburbs, the concentrations do not seem high enough to cause health consequences.

Our laboratory studies have also led us to an appreciation of the importance of the absorption/elimination characteristics of environmental chemicals. It is our view that the toxic properties of foreign chemicals ultimately depend on how well the body biotransforms and eliminates them and on how the body's immunological and other defense systems operate. It is clearly evident that more research is needed on the health effects of long-term, low level exposure to chemicals.

In summary, our work at PIEM indicates that a balanced scientific approach is necessary in the study of the health consequences of environmental risk factors. Any approach that limits its consideration to only one factor, like tobacco smoke, will hinder the generation and analysis of the epidemiological, pharmacokinetic, and toxicologic data needed to effectively study the causes of our nation's health problems.

STATEMENT OF LEON O. JACOBSON

I am Leon Orris Jacobson from The University of Chicago in Chicago where I continue as a physician-scientist emeritus and have served as Chairman of Medicine, Dean of Medicine and Biology and Director of the Argonne Cancer Research Hospital. In February 1942 I became responsible for the medical safety of the personnel of the Metallurgical Laboratory (the atomic bomb project) under the direction of Arthur Compton, Norman Hillberry, Enrico Fermi, Leon Szilard and many others who achieved the first self-sustaining chain reaction. Later I served as Associate Director and then Director of Medicine and biology of this enormously successful national program that brought the war abruptly to an end and in the aftermath provided the technology and the radioisotope tracers so enormously important to the revolutionary advances in biology and medicine that contribute an ever increasing momentum to our understanding of normal as well as disease processes.

I joined the Scientific Advisory Board of the Tobacco Industry Research Committee (now called the Council for Tobacco Research - U.S.A., Inc.) in 1954 when it was under the leadership of Clarence Cook Little. Dr. Little appeared before Congressional committees dealing with smoking and health in 1965 and 1969. The mandate of the Scientific Advisory Board was broad and simple, namely to sponsor independent research in the area of tobacco and health. The Scientific Advisory Board was and is today composed of individual scientists whose expertise covers those disciplines of the biomedical sciences that are essential to understanding the complexities of chronic diseases and their pathogenesis.

In spite of the successful elimination of many diseases caused by infectious agents, we are still groping for an understanding of the inexorable process of chronic diseases, such as arteriosclerosis and cancer. Are we in any better position today to fathom the challenging puzzle of cause and cure of these devastating medical problems?

We are becoming more aware of the fact that cancer, arteriosclerosis, and other chronic diseases are not the result of exposure to a single substance that has been inhaled or eaten. For example, arteriosclerosis is hastened by a concomitant hypertension, but occurs even in people who are of normal weight and without hypertension. What causes the arteriosclerosis that leads to coronary occlusions and strokes? Here we have a controversy that will be resolved only when each of the categories of research unravels the cascade of events that leads to death or disability. It would be disastrous, for example, to decide now that arteriosclerosis is primarily a problem relating to blood lipid control.

Cancer etiology and control have been intensively researched, especially in the past 75 years. We have learned much from the rapidly developing area of molecular biology which is driving us nearer and nearer to the ultimate goal of understanding the thousand and one continuous reactions in cell membranes, within cells and in organs and tissues that are required for maintaining the normal steady state. We are just beginning to understand the interrelationships of individual cells, tissues and organs such as the lung, kidney, and adrenals as well as the brain itself. Each of these body constituents is not an independently functioning entity, but rather part of a wonderful organism working in harmony in the healthy individual which quite often reverses or corrects abnormalities (or copying mistakes) that result spontanously or from injury or disease. Spontaneous mistakes in the process of cell division are inevitable because millions of cells must yield exact copies of the genetic information they possess to their progeny. The body has the ability to repair the strands of genetic material in which a mistake occurred in the reproduction process. If the repair is incomplete or fails then the newly formed cell might die or it may go on to develop a new kind of cell - a cancer cell.

How else might cancer occur? We know that selected mice can be inbred in a deliberate way (by 20 generation crosses) and a predictable percentage of their progeny will develop cancer. This is a very useful laboratory technique for testing carcinogenicity or cocarcinogenicity of a given chemical or other agent. Likewise, epidemiologists have found and are closely observing human "cancer families" in which the genetic role is clear. Thus, a full understanding of the complex process of carcinogenesis must consider not only abnormal inherited genes or chemical exposure of the genetic material of a cell but also the possible impact, involvement or participation of ionizing radiations and ultraviolet rays, viruses and many internal derangements.

In the laboratory animal, when studying tumor induction with a given chemical substance or virus, one has better control of the various factors which may play a role in carcinogenesis, but in human epidemiological studies it is very difficult to isolate one factor from another. Human beings are exposed to a variety of environmental impingements. They also have differing life styles involving various stresses, eating habits, drinking or smoking habits, as well as different genetic or constitutional backgrounds. We are seeking ways and there are promising leads in the search for the members of our population who may be susceptible or likely to develop cancer. We have found families with genetically transmitted susceptibility to emphysema. Likewise in the field of cancer, we are now looking for markers to identify people who might be susceptible.

Why have I stressed the importance of spontaneous, inherited or induced genetic aberration in the development of disease? Simply because many of our dread diseases seem to originate after a lesion occurs in a DNA strand, which may actually lead to the development of an individual cancer or become an inherited problem in successive generations. For example, molecular biology has already located the particular sequence in the double helix responsible for sickle cell anemia. Genetic engineering is a thing of the present that promises rapid progress.

Malignant cells carry unique and identifying molecules on their surfaces that are characteristic of the specific malignancy. With current techniques immunologists can develop Killer T lymphocytes that recognize and seek out the specific molecules of malignant cells and destroy the malignancy by attacking the surface molecules of the target malignant cells.

Many have heard of the hybridoma - a powerful tool that is revolutionizing biology. In the test tube a fusion of mouse tumor cells and antibody-forming cells results in a hybrid cell that can generate, perhaps immortally, large quantities of antibodies that will react with small peptide chains that are a part of a large protein. These hybridoma antibodies may also be used to treat tumors. This is an example of what genetic engineering can and is doing. We shall see in the next decade, and, in fact, we are already witness to a wide spectrum of genetic engineering procedures.

Even as I stand here at age 70, I am hopeful that we shall see the answers before the end of this century to many of the diseases that plague us. We are in the middle of vast and fruitful fields of molecular genetics. We can find the genetic abnormalities and we can even splice in new gene segments. We are literally swimming in new knowledge in immunology and only beginning to apply the fruits of these discoveries.

This has been a remarkable era of discovery in the area of peptide hormones which exert tremendous influence on many functions, including lung and cardiovascular. The future challenge is to see whether, and how, these peptides may play a role in the causation of chronic disease.

From these continuously emerging discoveries, only a few of which I have touched upon today, the synthesis of disparate findings occurs. It is this synthesis which heralds the understanding of the aging process. Only such understanding will lead to control of our most formidable and complicated diseases such as arteriosclerosis, heart diseases and cancer, as well as the whole category of other degenerative maladies.

STATEMENT OF LAWRENCE L. KUPPER, Ph.D.

1. Introduction

I am Lawrence L. Kupper, Ph.D., a biostatistician specializing in epidemiology and environmental health, currently Professor of Biostatistics in the School of Public Health, University of North Carolina at Chapel Hill. I have been a consultant to the U.S. Environmental Protection Agency and a member of the Instructional Staff of that Agency. I am currently doing research under a grant from the National Institute of Environmental Health Sciences.

Since 1967, I have been engaged in biostatistical research with particular reference to the analysis of epidemiological data on occupational health and cancer. I am an elected member of the Regional Committee of the Eastern North American Region of the Biometric Society and am a member of the American Statistical Association and the Society for Epidemiologic Research. I have published or presented approximately 100 scientific papers and am on the Editorial Board of the Journal of Chronic Diseases. My curriculum vitae and list of publications are attached.

My appearance at this hearing is voluntary and the opinions expressed are personal, not representing those of any organization.

2. Discussion

The widely-held belief that smoking "causes" lung cancer stems mainly from associations encountered in a wide variety of epidemiologic studies on human populations. Accordingly, it is important to discuss some of the limitations inherent in the use of epidemiologic research studies to assess the purported smoking-lung cancer relationship.

First, epidemiologic studies are generally observational rather than experimental. Epidemiologists are not able to conduct controlled experiments in laboratory-type settings where environmental conditions can be strictly monitored. Epidemiologists generally have no control over what their experimental units (i.e., human subjects) may be exposed to over a lifetime. A human being is exposed to a myriad of environmental impingements over a lifetime; these impingements take many forms (e.g., air pollution, diet, stress, exposure to occupational hazards, etc.), and they vary both in intensity and in the sequence in which they occur. Furthermore, the biological mechanisms by which such agents may act, either separately or in combination, to produce an adverse health effect are generally unknown.

If we want to compare two groups of individuals (e.g., smokers and non-smokers) with regard to their rates of development over time of some disease (e.g., lung cancer), how can we best deal with the fact that study populations are different with regard to genetic background, environmental impingements and so forth? We would be fairly content if we knew that these two groups were, on the average, equally susceptible to the disease before the suspected causal agent is introduced, regardless of previous exposure histories with respect to all other environmental impingements. One way to try to achieve such a desirable starting place for a follow-up study would be to randomly assign individuals to one of the two groups. This process of randomization represents an attempt to insure comparability, on the average, between the two groups with respect to both measured and unmeasured factors which vary from individual to individual and which may be risk factors for the disease under study. Although randomization is by no means a 100% guarantee that exact comparability will be achieved, it is certainly one reasonable way to balance the distribution of such factors (e.g., previous unknown exposures, genetic differences, diet, psychological and sociological factors, etc.) between the two groups.

Unfortunately, the technique of randomization is not available to the epidemiologist in most situations. For example, people who smoke have chosen to smoke, and people who do not smoke have chosen not to smoke. Thus, any epidemiologic study comparing smokers and non-smokers suffers from the basic flaw that the individuals have self-selected themselves into these two distinct groups. Thus, any observed differences between two such groups with respect to health outcomes may not, in fact, be the result of smoking, but may instead be due to other more basic factors (e.g., those of a genetic origin) which are necessarily different between the two groups because of the self-selection process itself. In other words, one can reasonably maintain the view that smokers are constitutionally different from non-smokers, and that such constitutional factors cause such individuals both to smoke and to develop lung cancer. Under this plausible hypothesis, then, smoking is an outcome variable just like lung cancer, being a manifestation of the constitutional factors uniquely possessed by people who choose to smoke.

Actually, there is considerable evidence that smokers are, indeed, a constitutionally different group of people from non-smokers (Burch, 1978). For example, many studies have shown that smokers differ, on the average, from non-smokers with respect to morphology, personality, and genetic markers. Studies of twins have shown that pairs of monozygotic twins (whether brought up together or apart) have much more similar smoking habits than do pairs of like - sex dizygotic twins, thus suggesting a genetic link with the tendency to smoke.

With regard to the relationship between genetic differences among individuals and differing levels of disease susceptibility, Janis and Kupper (1982) have recently demonstrated, by sophisticated statistical methodology, the existence (for U.S., and also for British, male lung cancer data) of a particular birth cohort (i.e., that group of individuals born around the end of the nineteenth century) which appears to be much more prone to develop lung cancer than do other birth cohorts born either before or after that time. The existence of such high-risk birth cohorts suggests that genetic, prenatal, and neonatal factors can be important determinants of subsequent disease susceptibility.

Based on all these findings, the self-selection bias problem is a very real one, which casts serious doubt on the ability of epidemiologic research studies to provide final answers regarding the causal relationship between smoking and lung cancer.

In addition, there are several other sources of bias that typically affect the validity of epidemiologic studies designed to address the smoking-lung cancer causality issue. These are:

1. Various studies (e.g., several of those referred to in the latest Surgeon General reports) involve particular sub-groups of individuals, which are in no way representative of the general population and which are excellent illustrations of the self-selection syndrome. Examples of such studies are the British male doctor study of Doll and Hill (1964), and the studies of Seventh Day Adventists (e.g., see Lemon and Walden, 1966) and Mormons (see Enstrom, 1975). It is well known that doctors differ from the average person in very many respects; and, members of religious sects which prohibit the use of alcohol and tobacco generally are much different personality-wise from the general population. Clearly, findings regarding such unique sub-groups cannot be applied to the general population.

2. It is well documented that the diagnosis of lung cancer is a difficult clinical problem and that, in fact, the diagnostic criteria and associated diagnostic error rates have changed considerably over the years. Such changes can undermine the validity of associations between smoking and lung cancer reported in various epidemiologic studies.

3. A more subtle form of bias, known as detection bias, has been discussed by Feinstein and Wells (1974), specifically with regard to the smoking-lung cancer controversy. Generally speaking, such a bias can arise when the knowledge of exposure or non-exposure to an agent alters the objectivity of the scientist. For example, an M.D. who knows a person is a heavy smoker may be more on the lookout for lung cancer in that person than he would be for a light smoker or a non-smoker. This form of detection bias would obviously tend to exaggerate the smoking-lung cancer association, as has been demonstrated by Feinstein and Wells with real-life data.

4. It is well-known that there is considerable inaccuracy associated with the use of exposure information based on questionnaires and interviews with next-of-kin to assess

habits of deceased relatives with regard to smoking or drinking, even though this is often the only source of such information in many epidemiologic studies.

5. With regard to the control of confounding factors when studying the smoking-lung cancer controversy, probably the single most common and important factor is age. And, unfortunately, the effect of age on cancer causation processes is little understood. Eminent cancer researchers believe that the aging process, in and of itself, is causally associated with the development of cancer (e.g., see Blumental, 1978).

6. There are two main types of epidemiologic studies: the follow-up study and the case-control study. Such studies have reported an association between smoking and lung cancer. However, both kinds of studies can seriously suffer from the presence of biases. The case-control study is probably the epidemiologic study design most susceptible to bias; in fact, an entire issue of the Journal of Chronic Diseases (1979, Volume 32, No. 1/2) is devoted exclusively to discussing the biases and problems associated with case-control study research. The follow-up study, in addition to the general list of biases already discussed, is subject to a special form of selection bias, namely, loss-to-follow-up; in particular, we are refer-

ring to individuals who, although initially enrolled in the study at the start of the follow-up study period, are lost during the subsequent follow-up period because of migration, death from other causes, refusal to continue participation, etc. Such losses are characteristic of all long-term follow-up studies (e.g., like those designed to study the smoking-lung cancer issue), and can seriously bias the conclusions from such studies. Greenland (1977) has illustrated the seriousness of this problem with some well-chosen numerical examples.

3. Conclusions

The belief that smoking is a cause of lung cancer can be questioned in light of the documented sources of bias attendant with epidemiologic studies of the smoking-lung cancer relationship.

Statistical associations between smoking and lung cancer, as reported in the various studies described in the Surgeon General reports, have been interpreted to mean that a causal relationship does exist. In the absence of well-designed animal and laboratory studies elucidating the meaning of these reported associations, such a quantum jump from association to causality is invalid. Indeed, the self-selection bias itself (not to mention all the other possible sources of error) is sufficient to cast doubt on the causality claim.

The main prospective (i.e., follow-up) studies concerning smoking and its relationship to mortality and morbidity from certain diseases (as discussed in the latest Surgeon General reports) do not address the issue of causality. The claim that the conclusions reached are "impressively uniform and consistent" does not mean that a causal relationship has been established, but only that approximately the same observed associations keep appearing. In fact, the biases inherent in these studies preclude the right to claim that causality has been demonstrated. Actually, one could argue that the same associations keep appearing because the same biases are present in each study.

The fact that so many studies have produced a positive association between smoking and lung cancer has led many people to make the false conclusion that "quantity means quality". The truth of the matter is that repeatability (i.e., the ability to produce the same estimated association) does not imply accuracy (i.e., the ability to produce a valid estimate of the true association). In this regard, one study free from all bias and producing a valid measure of the true smoking-lung cancer association is worth more than a thousand biased studies, all of which provide the same distorted estimate of the true association.

In summary, based on the currently available information pertinent to the smoking-lung cancer causality issue, I am of the opinion that a causal relationship between smoking and lung cancer has not been scientifically demonstrated.

STATEMENT OF HIRAM THOMAS LANGSTON, M.D.
CLINICAL PROFESSOR OF SURGERY (EMERITUS)
NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

I am Hiram Thomas Langston, a thoracic surgeon in private practice in the Chicago area. I am the Chairman of the Department of Surgery at St. Joseph's Hospital in Chicago, and a Clinical Professor of Surgery (Emeritus) at Northwestern University Medical School.

I appreciate this opportunity to present my views on H.R. Bill 4957/Senate Bill 1929. I am concerned, however, with several claims made in them, especially those which endorse the hypothesis that smoking is the main cause of lung cancer.

Smoking has been said to be responsible for causing an unbelievable array of illnesses, including lung cancer. Much of the support for these accusations comes from research that is basically statistical.

Since the early 1960's, I have read most of the scientific literature on smoking and lung cancer. In my capacity as a thoracic surgeon, I do not feel qualified to respond directly to the reported statistical associations between smoking and cancer. However, I must respond to the interpretation of these associations as causal because it is inconsistent with the clinical realities of the disease that I have observed for the past forty years. Adopting the old adage "it is the exception that proves (tests) the rule," I identified certain very pertinent "exceptions by observing firsthand the clinical behavior of lung cancer. These exceptions cast doubt upon the validity of the hypothesis that smoking causes lung cancer.

1. Inhaled cigarette smoke is equally distributed in both lungs. Why, then, as the data show, and as I have observed in my own practice, do lung cancers very rarely appear simultaneously in both lungs? The answer is not known, but this phenomenon is inconsistent with the smoking causation hypothesis.

It is of further interest to note that the vast majority of people who have been successfully treated for one malignant tumor in the lung do not develop subsequent lung tumors.

2. Cancer rarely occurs in the trachea (windpipe). The trachea is exposed to more tobacco smoke than are the lungs, because all the smoke is inhaled and exhaled through it. Also, the material deposited in the mucous lining of the air passages exits through the trachea.

The trachea is anatomically, embryologically and physiologically identical to the rest of the bronchial airway. Therefore, if cigarette smoke were a cause of lung cancer, one would also expect to see a large number of tracheal cancers. The fact is, however, that tracheal cancer continues to be an extremely rare disease.

3. Cancer of the larynx or voice box is also statistically linked with smoking. Because cigarette smoke passes through the larynx on its way to the lung, the larynx is exposed to at least the same concentration of smoke as are the lungs. Were the smoking-causation hypothesis valid, one would expect to see a rise in laryngeal cancer similar to the rise in lung cancer. Yet, the data show that there has been little change in the incidence of laryngeal cancer over the past decades.

4. I regard with a certain amount of suspicion the view that we are in the midst of a lung cancer epidemic because of cigarette smoking. Any discussion of this "epidemic" must take into account two frequently overlooked clinical factors that have had a tremendous effect on the reliability of reported lung cancer rates: (1) diagnostic techniques and (2) official certification of cause of death.

Even in the time span of my own practice, I have seen remarkable changes in our ability to diagnose lung cancer. When one considers that even diagnostic x-rays were not readily available a scant decade or two before I started practicing, it is hardly surprising that our ability to detect lung cancer has increased dramatically. And as that ability has increased, so naturally have the reported lung cancer rates.

Earlier in this century, physicians may have failed to diagnose lung cancer, resulting in rates lower than the actual incidence of the disease. Thus, when these unrealistically low rates are compared with rates for later periods when diagnostic tools were gradually becoming available, one would obtain a false impression of the real increase.

The other factor I believe to be important in evaluating whether there is a lung cancer epidemic is the accuracy of death certificate information. Death certificates are the sources for calculating death rates, but unfortunately, information in them is extremely unreliable. Most layman assume that death certificates accurately reflect the cause of death, but in many cases they do not. Coroners and non-treating physicians sign many death certificates, and they may have little or no relevant information about the actual cause of death. Even treating physicians make mistakes. That is why I have refused to consider in my own population studies any case as lung cancer unless there was microscopic confirmation of the diagnosis. Many cases lack that confirmation.

This is not to say that there has been no increase in lung cancer. I am quite convinced that a portion of it is real. I am equally convinced, however, that we have not yet identified its cause or causes.

5. In my review of the literature, I have seen the argument that the epidemiological studies show a dose-response

relationship, that is, the greater the exposure to cigarette smoke, the greater the risk of developing lung cancer. Whereas I cannot directly challenge the statistical analyses used to obtain these associations, I have been able to consider another aspect of "dose-response" -- age at diagnosis. The age at diagnosis of lung cancer does not seem to be related to the age at which a person started smoking, nor how long he smoked, nor even the number of cigarettes he smoked per day. I have observed this in my own patients, and indeed I have found it to be confirmed in the literature.

6. Age-specific lung cancer death rates almost always have a special pattern. In most series of lung cancer patients, the greatest rates occur in the 50 to 70 year age group, with a peak at 60 years. The literature also reveals that a certain generation (those born before the turn of the century) may have higher lung cancer rates than other generations.

Intrigued by these findings and the possibilities that they suggested, I reviewed approximately 4,000 lung cancer cases spanning 30 years at the Veteran's Administration Hospital in Hines, Illinois. All cases carried the diagnosis of lung cancer supported by microscopic evidence.

I found that (1) the generation born between 1890 and 1900 contributed the largest number of cases; (2) if this trend continued, this generation would fade from prominence due to old age; (3) the younger generations did not appear to be replacing this generation in cancer production. Given these points, I predicted that the number of lung cancer cases at the VA Hospital in Hines would decrease.

In a subsequent investigation of cases through 1978, I discovered that the contribution of the generation which had earlier produced the greatest number of cancers at Hines had in fact decreased significantly. In addition, the total number of cases at Hines in the period 1968 through 1978 had dropped approximately 17%. This seems to be a rather significant change which supported my earlier predictions. This observation now spans 45 years and encompasses approximately 5500 cases.

7. Lung cancer is a dynamic disease in the sense that its occurrence patterns and clinical appearance (cell type) are ever changing. For example, there appears to have been a decline in the rate of increase of lung cancer. Indeed, lung cancer incidence may have, in fact, crested. Other investigators, including some who believe that smoking causes lung cancer, seem to concur with this observation. For instance, in his address before the Health Congress in England in 1977, Sir Richard Doll said "it is encouraging to find that the total death rate from lung cancer in men decreased in 1975 albeit very slightly, for the first time in 50 years."

Perhaps what we are seeing in the case of lung cancer is what is called the "natural history" of this disease. Natural history has been succinctly described by a British thoracic surgeon as the "long drawn out process of the development and the decline of an individual disease."

If you have trouble accepting the idea that a spontaneous decline in lung cancer can occur, I remind you of the documented decline in stomach cancer. The spontaneous decline in stomach cancer over the years is a decline for which no convincing explanation has been offered. Improvements in nutrition or food storage, or diagnostic refinements, or changes in the general health of the population do not adequately explain these changes.

What explains the changes in lung cancer rates? As Dr. Belcher has pointed out, the decline in lung cancer's rate of increase started before changes in the cigarette occurred. Is this simply another example of the poorly understood natural history of a disease? Clearly, no simple explanation for these lung cancer changes appears to be forthcoming.

Many important questions about cancer causation remain unanswered. For example, precise causal mechanisms have not been identified. Many theories have been proposed, but none have won universal acceptance.

I do not agree that cigarette smoking is the major cause of lung cancer, because I believe very strongly that we do not know the cause or causes of cancer of the lung. Charges that smoking causes lung cancer are so familiar that very few people may realize that there is strong evidence to the contrary. I find that evidence to be persuasive. In my estimation, the smoking hypothesis is an oversimplification. I therefore cannot support legislation such as these two bills based upon such a questionable hypothesis.

Hiram T. Langston, M.D.

The Alleged Costs of Cigarette Smoking

S. C. Littlechild, B.Com. Ph.D.
Professor of Commerce
Head of Department
Department of Industrial Economics
and Business Studies
Faculty of Commerce and Social Science
The University of Birmingham

House of Representatives Bill No. HR4957, introduced by Mr. Waxman on November 12th 1981 contains on page 2 the following passage :

> "(8) it is estimated that cigarette smoking related deaths and disabilities result in $25,800,000,000 annually in lost productivity to the United States economy and $13,600,000,000 in medical costs."

As an applied economist with experience in cost-benefit analysis, I have been interested in this and related issues for several years. As I indicate below, there are numerous problems in making statements of the kind just quoted. In my experience, most of these statements are based upon inadequate economic analysis and unreliable statistical data. As a result, they tend to be seriously misleading, therefore it seems worthwhile to set out briefly some of these difficulties involved.

1. It is by no means established that cigarette smoking is a cause of illness and death as claimed in items (1) to (6) of HR4957. Since this is not my area of expertise I shall not dwell on it further. But the point to establish is that the reliability of any cost calculations cannot be any greater than the reliability of the underlying medical diagnoses. If the medical experts cannot agree on the effects of smoking, either in general or in specific instances, it follows that there cannot be agreement on the costs of smoking. Indeed, if one takes the view that smoking is statistically associated with certain illnesses but is not the cause of those illnesses, then the value of lost productivity and medical costs properly attributable to cigarette smoking is zero.

2. Even if cigarette smoking is claimed to be a cause of illness and premature death, the extent of any such contribution remains uncertain. There are inevitably a number of contributory factors associated with the incurring, severity and duration of any illness and with the timing of death. For example, personal habits of diet and exercise, as well as personality factors, influence the onset and severity of certain illnesses, which in turn affect the number of days off work or spent under medical care. Even if smoking were

a contributing factor to illness and premature death, it would be quite inaccurate to attribute to smoking the whole of the associated costs. But exactly what proportion ought to be attributed is unclear, partly because the extent of any contribution of smoking is uncertain, and partly because there is no basis in pure logic for attributing joint effects among the various possibly contributory factors.

To illustrate, specific examples of this point are found in items (5) and (6) of HR4957, which claim that "women who take birth control pills and smoke are more likely to suffer a heart attack or stroke" and that "certain occupational hazards in conjunction with an individual's smoking increase substantially the risk of disease and death". The first difficulty is an empirical/medical one : how far is it possible to identify and measure accurately the separate effects, if any, of pills and smoking, or occupational hazards and smoking ? The second difficulty is one of principle : how far should any combined effects of pills and smoking be attributed to smoking rather than to taking birth control pills ? If, to take a hypothetical case for illustrative purposes, it were established that taking a specific pill and smoking had no effect separately, but had a specific effect when combined, it would be quite arbitrary to attribute the effect (and its consequent costs) to smoking alone.

3. People engage in an enormous variety of activities some of which may result in accidents, illness or death, thereby reducing productivity and generating medical costs. Dramatic recreational examples include skiing, mountain-climbing, potholing (cave exploring), and skydiving; more mundane examples include travelling by car or plane, or over-indulgence in food, drink, physical exercise, sunbathing, nightlife and so on. All these activities generate costs in some cases presumably of substantial magnitude, but while these costs are to be regretted, it is recognised that they do not constitute the whole of the story. There are corresponding benefits to be taken into account, which in the eyes of those who engage in these activities more than compensate for the costs incurred. Calculations of the purported costs of cigarette smoking generally take no account of the benefits perceived by smokers. In this respect, cigarette smoking is being singled out and treated differently, for reasons that are not made clear.

4. Calculations of the claimed costs of smoking frequently assume that if people were not affected by illnesses allegedly caused by smoking they would otherwise be perfectly healthy.

This, of course, is not the case : They are typically affected by a variety of illnesses. The relevant loss in productivity is thus related not to the total absence from work experienced by smokers, but to the incremental absence, if any, that can properly be attributed to smoking. This would be naturally much lower.

5. Similarly, studies often calculate the total medical costs incurred by smokers. This is incorrect : the appropriate calculation is the incremental cost, if any, attributable to smoking. In particular, it needs to be taken into account that nonsmokers who live longer tend to generate very substantial medical costs in old age. Indeed, several studies by economists sympathetic to the anti-smoking cause have concluded that the net medical cost of smoking is negative - that is, the lifetime costs of treating smokers are in fact less than the lifetime costs of treating non-smokers.

6. Finally, it might be thought that the costs of smoking are incurred not by the smoker but by the rest of society, so that smokers are effectively subsidised by non-smokers. This is far from the case. Any lost productivity due to absenteeism or ill-health is probably reflected in lower wages, and medical costs are frequently born by the patient (possibly in the form of higher insurance premiums). But any such costs are far outweighed by the heavy taxes on tobacco products. There is no doubt that, on balance, there is a transfer of income from smokers to the rest of society.

For these six main reasons, calculations of the claimed costs of smoking are fraught with difficulties, and those that I have studied have generally proved inadequate and misleading. It is possible that the figures embodied in HR4957 are based upon new and more acceptable calculations, but in the absence of such evidence I am inclined to view them with suspicion.

Statement of Eleanor J. Macdonald
Professor Emeritus of Epidemiology
Department of Cancer Prevention
University of Texas System Cancer Center
M. D. Anderson Hospital and Tumor Institute, Houston, Texas

My name is Eleanor Macdonald, Professor Emeritus of Epidemiology at the University of Texas System Cancer Center, M.D. Anderson Hospital and Tumor Institute, Houston. I have worked in the field of epidemiology for over 40 years in three state programs in Massachusetts, Connecticut and Texas; in fact, I established the first state cancer registry in the U. S. From 1948 through 1974, I chaired the Department of Epidemiology at the University of Texas System Cancer Center. I have authored or coauthored about 150 publications, most of which deal with different phases of cancer epidemiology, and I remain actively involved in a number of ongoing research studies in epidemiology. I am also the editor of the Epidemiology, Statistics and Cancer Control section of the Yearbook of Cancer.

I share the concerns of this Committee regarding cancer morbidity and mortality, and I encourage legitimate efforts to control this disease. However, as a scientist who has dedicated her professional career to a careful study of cancer epidemiology, I am appalled at the belief implicit in H.R. 4957, H.R. 5653, and S. 1929 that Congress can legislate scientific fact. I urge this august body of well-intentioned legislators to avoid putting itself in such an untenable position.

These bills are a misdirection of governmental energy and purpose because of their narrow focus on smoking as the major cause of lung cancer. We do not, in fact, know the cause or causes of lung cancer and it is not in the best interest of either science or government to take a simplistic approach to the task of identifying them. There are obviously many factors in cancer causation that we have not yet begun to understand. For example, the depth of our uncertainty is still far too great regarding the effects of nutrition, work history, life style, ancestry and environment.

One of the main reasons that I disagree with these bills' findings regarding smoking and cancer stems from my knowledge of the primary type of evidence on which these judgments are based, i.e., epidemiological. As an epidemiologist who has conducted large-scale population studies, and who has reviewed the data of vast numbers of research reports, I am perhaps more aware than many scientists -- and certainly more aware than most laymen -- of the limitations of this type of data. Congress must understand that epidemiology is not an exact science, and no amount of wishing can make it so. We cannot turn humans into animals directed at will toward various exposures.

We cannot send all death certificates to one infallible pathologist for correction and confirmation. We cannot force all individuals supplying the raw data to epidemiologists, or even all epidemiologists themselves, to demand the type of precision necessary in data gathering and review to insure that the quality of the data meets high scientific standards. Consequently, judgments about chronic disease causation based on epidemiological findings must be highly tentative; they do not enjoy the absolute certainty implied in the findings of these three bills.

Mortality data are frequently used by epidemiologists to study disease trends, often without full awareness of inherent weaknesses in these data. Mortality rates are obtained from information on death certificates furnished by physicians or local health officials. Although I am certain that every effort is generally made to fill these out conscientiously, errors do occur -- in diagnosis as well as in recording of the data.

In addition, other exogenous influences can cause profound fluctuations in mortality data over the years that can easily be misinterpreted as changing disease trends. For example, the introductions of each of the seven revisions of the International Lists of Diseases and Causes of Death (ICD) have had effects, generally improvements, on the classification of diseases. With each revision, however, the continuity of the disease trends has been broken and in some cases seriously distorted. Sadly enough, even though it has taken us centuries to attain the classification system that we now have, it is still imperfect.

The possibility of shifting classifications bringing about a rise or fall in certain causes of death makes some investigators question the value of any effort to compile statistics on causes of death. For example, anyone attempting to trace the patterns of lung cancer during the twentieth century must remember that the 1948 ICD revision, first applied in 1949, introduced a classification separating primary cancer of the lung from metastatic cancer of the lung (coded thereafter as "unspecified as to primary or secondary). For two years, there was a sharp reduction in the reported primary lung cancer rate, and then a rise in both the primary and unspecified rates. How long did it take for the new classification to catch on? How accurately did physicians distinguish between primary and secondary? When epidemiologists study twentieth century lung cancer trends, do they include or exclude the unspecified and secondary cancers? We cannot be sure about the answers to these questions.

The 1965 ICD revision, adopted in 1968, seriously complicated the situation by deleting the unspecified category. This revision effectively combined primary and unspecified lung cancer, thereby removing a needed safeguard for accuracy. Primary and secondary lung cancer are separate disease entities, with quite possibly distinctly different causes. Since ten percent of all cancers spread to the lung, and since, for nearly twenty years, more deaths were coded in the unspecified than in the primary category, the combination of those two categories seriously confused the lung cancer data. The damage done to epidemiological investigations of lung cancer by the 1965 classification change cannot be underestimated. In the ninth revision (1980), greater confusion was added by placing the bronchus and lung unspecified as the ninth subdivision of primary lung cancer (162.9). Death certificates seldom list more than a three-digit number, such as 162. We simply cannot separate out from the mortality data the specific information we need to study lung cancer scientifically.

Another aspect of the problem of changing disease classifications can be found by comparing trends in stomach and lung cancer. In the first half of this century, the phrase "question of gastric cancer" was used frequently on death certificates when a cachectic patient died within days or hours of first summoning a physician. It is fascinating to see that in

47 states, within two years of the introduction of the 1948 revision, the reported gastric cancer mortality rates dropped as suddenly as the unspecified lung cancer ones rose. Thus, the two changes may in part represent a shift of questionable diagnosis from the difficult to diagnose stomach cancer to the equally difficult to diagnose lung cancer classification.

Besides studying anomalies that have developed in overall lung cancer trends, I have also examined trends in the reported lung cancer mortality rates for women in this century. For years, those individuals who believed that smoking caused lung cancer focused almost solely on male lung cancer, and generally tended to ignore female lung cancer. In recent years, however, more attention has been given to female lung cancer because of reports of an increase in female lung cancer and the claim that increased cigarette smoking was responsible. I do not accept this interpretation because I have serious scientific reservations about both the accuracy of the underlying data and the conclusions drawn from it.

First of all, we simply do not have reliable data on smoking prevalence in women -- or in men, for that matter. The best information we have is from the National Health Survey reports, yet the 1978 report, for example, has a standard error of 30 percent. That percentage of error is too high to provide a basis for drawing definitive conclusions.

Secondly, I have doubts about whether lung cancer in women has actually increased dramatically. As I indicated earlier, national mortality data may not be reliable. If we assume for the sake of argument, however, that they are reliable, we can still question the argument that female lung cancer has increased sharply in recent years. Although the age-adjusted U.S. death rates from respiratory cancer in white females show a consistent increase from 1953 through 1975, the rate of this increase has varied over this period. A smoothed year-to-year variation in the rate of increase -- the slope -- indicates that a sharp rise began around 1960, but leveled off around 1970. Although the rate of increase has begun to rise again, it is not as steep an ascent as in the 1960's.

This is important because the slope gives a predictive picture of the overall mortality trend. And the slope that I see for female lung cancer suggests a stabilization. It may even predict a decline in lung cancer mortality in the coming years.

Other factors must also be considered in an analysis of the apparent increase in female lung cancer. One such factor, inaccurate reporting, appears to have played a predominant role.

My analysis of both reporting techniques and the underlying data suggests that lung cancer in women was underreported in the past and is being overreported now.

Underreporting may have occurred primarily because of three clinical factors: 1) less clinical interest in female lung cancer, 2) inadequate diagnostic tools, and 3) clinical misdiagnosis of lung cancer as tuberculosis or other respiratory diseases. First of all, for many years, clinicians tended to believe that women were less likely than men to develop lung cancer, and that belief could have affected their diagnoses. Second, physicians were less capable of detecting lung cancer, because they simply did not have adequate means of discovering the presence of a cancer. Third, mainly as a consequence of these limitations, physicians may have confused lung cancer with other respiratory diseases, particularly tuberculosis. In other words, they may have diagnosed tuberculosis when in fact the patient had lung cancer.

The introduction of antibiotics and other drugs led to a sharp decline in deaths from tuberculosis and other infectious diseases. With isoniazide and these other drugs at their disposal, physicians could suddenly separate the lung cancer patients from the tuberculosis patients, because the TB patients generally improved after drug therapy whereas the cancer patients did not. The abrupt drop in tuberculosis mortality rates after the introduction of isoniazide in 1952, and the nearly simultaneous increase in reported lung cancer rates, provide substantial support for my belief that these diseases were often confused clinically. Therefore, as these drugs became increasingly available, underreporting of female lung cancer became less common.

Unfortunately, there is evidence which suggests that today lung cancer in women is overreported. This may occur in one of two ways: (1) clinicians are not always able to distinguish between primary and secondary lung cancer. As discussed earlier, the distinction is of obvious importance for a factual count of primary lung cancer cases. The only truly effective way to determine whether lung cancer is primary or secondary is through autopsy, and few cases of cancer deaths in this country are autopsied. In general, the autopsy rate is about 12 percent. When one considers that this percentage includes all the accidental and violent deaths that must be autopsied, one understands just how low the autopsy rate for lung cancer may be. This issue becomes even more crucial because primary lung cancer is one of the more difficult diagnoses to establish clinically. (2) The current ICD classification combines primary and unspecified lung cancer into one category. Even if the clinician could correctly distinguish primary from unspecified lung cancer, the effort would be negated by classification methods. Thus, both primary and unspecified lung cancers are combined in the reports that have described increases in lung cancer in women.

Therefore, changes in diagnostic techniques and changes in disease nomenclature are very important because, speaking from the epidemiologists' point of view, we cannot be sure that the reported trends in lung cancer among women accurately reflect the true incidence of the disease.

Further support for my skepticism regarding an identification of cigarette smoking as the primary cause of lung cancer stems from my own research investigations of this disease. In one, a study of lung cancer in Spanish surnamed and Anglo women in El Paso, we found a high incidence of the disease in the Spanish surnamed, 64 percent of whom were smokers. Although the same percentage of the Anglo women also smoked, their rate of lung cancer was half that of the Spanish surnamed. We concluded that we had to rule out smoking as the significant factor. Instead, the most important factor in the Spanish surnamed women's lung cancer was their residence from birth to adulthood in adobe houses. These are solidly built, nearly airtight and poorly ventilated structures heated mostly by wood fires, which exposed the women to known carcinogens.

In a much larger study, my colleagues and I examined the relationship of environmental and ethnic factors to cancer mortality by site for every state and region of the U. S. for a twenty year period. We began with a very large number of variables or factors, from which we selected 12, among them cigarette consumption, intake of different kinds of alcoholic beverages, ethnic background, pollution, income, temperature, etc. We used these to derive sophisticated statistical models of the geographic patterns of cancer mortality in the U. S. These models were an attempt to describe by statistical equations the relationships between certain factors and cancer mortality.

We found that all the factors used in the study accounted for only 36 percent of the geographic variation in white female death rates from cancer of the bronchus and lung specified as primary. The best model -- which eliminated the unimportant factors and brought into focus the important ones -- accounted for 29 percent. (In contrast, the models accounted for 89.5 percent of the variation in cancer of the breast.) This indicates that unknown but crucial factors were not included in the variable pool from which the model for primary lung cancer was derived. Cigarette consumption, which was included, turned out not to be a factor.

We also discovered a warping in the basic data dividing primary lung cancer from the unspecified category of lung cancer -- for both white females and white males. This is important because it shows we can't be sure lung cancer rates, as reported, present a factual picture.

There are other important considerations required in a balanced discussion of lung cancer in women. For instance, women in increasing numbers have, over the last 35 years, left the jobs they have traditionally performed and have entered work areas formerly reserved for men. Women are now exposed to the same community and work environments as men, and there are many unknown carcinogenic potentials in these environments.

The tremendous variety of toxic and carcinogenic airborne chemicals produced by industry and released into indoor and outdoor environments, in our homes and our workplaces, suggest a set of factors which may be important in lung cancer etiology. In 1976, I published a 30 year study which examined the possible influence of the petro-chemical industry exposures in Houston upon lung cancer mortality, heart diseases, and other respiratory diseases. In certain areas clustered around industrial facilities, mortality rates from respiratory cancer and other diseases were found to be very high -- so high, in fact, that deaths from respiratory diseases nearly doubled over the last 15 year period. Outside the industrial "pathway," mortality rates had remained stable for twenty years. A similar finding was made by Clemmesen in an industrial city in Denmark. These epidemiological findings are strongly suggestive of the fact that industrial exposure factors underlie the etiology of lung cancer. I might add that I saw no evidence that cigarette smoking could explain the peculiar disease patterns I found in Houston.

Our increasing ability to identify industrial carcinogens has opened many avenues of study and emphasized the importance of industrial environments. Control of these carcinogens at their source offers the most positive potential for prevention of lung cancer.

To date, epidemiological studies of lung cancer have not generally included adequate information about occupational exposures. Frequently, an epidemiologist may get little more than a brief indication of occupation -- such as, "construction worker." This tells us nothing about that person's actual exposure. At the M. D. Anderson, one of the major centers in the U. S. for the study and treatment of cancer, records are even less informative: the vast majority of cancer patients' records simply list "retired" as the occupation. How helpful, then, can we expect the records of private practitioners and community hospitals to be? Even when attempts are made to elicit full occupational exposure history, the patient's knowledge and/or memory of previous exposure will undoubtedly be faulty and incomplete. I seriously doubt if the typical industrial worker has an exact record of the hazardous substances, much less the amounts, to which he may have been exposed over a 30-40 year time period. And his wife, who may well describe her occupation as housewife, would undoubtedly know even less about the substances she cleaned from his work clothes all those years.

Still other important areas of research, besides the above mentioned, are emerging in our study of the puzzle of cancer causation. Scientists are beginning to recognize the possible roles played by nutritional imbalances in the etiology of cancer. Although this discipline is still in its infancy and conclusions do not appear to be immediately forthcoming, this new area of research does point to the fact that there exist many scientific unknowns in the study of cancer causation.

From my own studies and from a considerable amount of other research reported in the literature, it is therefore apparent that calling cigarette smoking the single major causative factor in female lung cancer is an oversimplification of a very complex epidemiological problem.

Conclusion

There is a concept widely accepted by public health schools that if many studies, in themselves not based on definable populations, all arrive at the same conclusion, then that conclusion must be valid. This concept disregards the fact that the same intrinsic error in method might produce the same result.

In the first and subsequent Surgeon General's reports, one of the main sets of supportive data was based on the survey of the American Cancer Society. The inadequacy of this survey was recognized and critically analyzed by many of the foremost statisticians and epidemiologists at the time, including Sir Ronald Fisher, the father of statistical methods as we now practice them, Dr. Joe Berkson of the Mayo Clinic, who devised the method of reporting survivals we all use, Professor K. A. Brownlee, for the American Statistical Association, Dr. Donald Mainland, physician and teacher of medical statistics, and numbers of others.

In the Cancer Society study, thousands of women volunteers asked of their acquaintances many questions about their personal habits. No one questions the sincerity of the women, but no business would consider basing its sales policy on the results of such a study. The proportion of smokers interviewed bore no resemblance to the proportion of smokers in the population, which demonstrated that the women surveyors went out to find smokers. Twenty-five states were not in the study. It was largely an urban New York, New Jersey population, of upper middle class people.

An enormous literature has grown up on the subject of smoking. But the bulk of the evidence is statistical. In no single instance has cancer of the lung been produced in any meaningful animal experiment using tobacco smoke inhalation, even though millions of dollars have been spent in the effort.

In all my years as a cancer epidemiologist, I have seen many theories st[illegible] and debated. But only in the instance of the smoking theory has the theory itself become sacrosanct. Any of the many valid scientific observations which refute the theory are discounted as bizarre. Many a doctorate on human psychology will be earned in the future, when the actual cause of lung cancer becomes known, which will attempt to explain the intensity of the promotion of this unproved theory.

Science, by explaining one fragment after another of genetic, biochemical, somatic, behavioral, environmental, and nutritional information, is building gradually the structure, which, upon completion, will help us understand the genesis of cancer. It is becoming increasingly clear that there is no single, simplistic answer to the question of what causes respiratory cancer. It is hardly in the best interest of either science or government to create the illusion that an attack on a single lifestyle factor will provide the solution for such a complex problem. The proposed legislation creates this illusion and should not be passed into law.

STATEMENT OF JOHN E. O'TOOLE, CHAIRMAN, FOOTE, CONE & BELDING COMMUNICATIONS, INC.

Mr. Chairman, members of the subcommittee. Thank you for allowing me to appear today in this consideration of H.R. 5653.

My name is John E. O'Toole. I am chairman of the board of Foote, Cone & Belding Communications, Inc., the fourth largest U.S. advertising agency -- ninth largest worldwide -- with 34 offices in 19 countries. Our New York office works on special product assignments for Lorillard and one of our Chicago accounts is Brown & Williamson International Tobacco. Foote, Cone & Belding is the successor agency to Lord & Thomas, one of the oldest U.S. advertising agencies, which for many, many years from the turn of the century worked with the American Tobacco Company.

Before I address the proposed legislation, it would seem appropriate to discuss for a moment the theory and purpose of advertising. Eighty years ago the generally accepted definition, was "keeping your name before the public." In 1904, a bright young employee of Lord & Thomas, Albert Lasker, and a former Royal Canadian Mounted Policeman named John F. Kennedy who became a Lord & Thomas copywriter, redefined the mission of advertising in a way that was to affect our industry and indeed all of those industries offering the public goods and services from that day to this.

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Advertising, as Lasker and Kennedy defined it, is "salesmanship in print." This realization changed the course of advertising completely. It added the concept of persuasion, the prime role of the salesman, thus producing a creative explosion that made the advertising industry a potent force in the American economy and taught businessmen throughout the world how advertising could move their products and stimulate the economies of their own cities and nations.

Advertising, then, is salesmanship, functioning in the paid space and time of mass media. It is based on the information with which a salesman must be equipped to make a person-to-person sale. In the old days it might have persuaded a consumer to buy a new type of product, as Albert Lasker did with American Tobacco's new blended cigarette. Today, however, cigarette advertising persuades the smoker to try another brand. It does so on the basis of what our research tells us smokers are seeking in a cigarette -- low tar, perhaps, or flavor, or the right balance of each.

If I, as a smoker, try a pack of cigarettes not usually my brand, because a magazine ad has persuaded me that it is richer and fuller in flavor than those I now smoke, I do not have to buy a second, third or fourth pack if I find the product not up to my old one. Through advertising I make an informed choice to try a product.

It has been said that the new rotating labels proposed by H.R. 5653 will enable the smoker to make a more informed choice. And here we come to the crux of my testimony here today. Say that it is decided that a brand of cigarettes selected to carry what I will call the pregnancy warning is my brand. Say that among the brands that must under the system to be created by the Federal Trade Commission carry for the first year what I shall call the heart disease warning is the favorite of my next-door neighbor and friend. Now I am not of a mind to bear a child. Much as my wife and I love our two daughters, we do not really care to add another child to our family now. And my neighbor, who has the lowest blood pressure on the block, knows of no one in his family for generations back with any hint of heart disease. If I read on my pack everty time I reach for a cigarette that smoking and pregnancy don't mix -- if my neighbor makes mental note that the Congress has mandated the heart warning every time he picks up his pack -- are we being informed of anything that is pertinent to us as smokers? Aren't we and millions of smokers to whom the warnings on their favorite brands have little or no relevance in effect being misled? If, conversely, I am worried about heart disease because I know of a family tendency toward coronary occlusion, need I shop for the brand whose advertising does not mention diseases of the heart? Should the woman, who is especially conscious of cancer because her mother, or best friend, or a person with whom she works have all had cancer, search for a brand that speaks only of heart disease?

When the Congress decided in 1965 that cigarettes should be labelled as potentially dangerous -- and when the warning label then appeared in advertising in 1972 -- it was, as the 1965 legislation states, that "the public may be adequately informed that cigarette smoking may be hazardous to health." From all accounts, except the FTC's, the label on packs and in advertising has been effective in spreading that word. Would it not be, in effect, deceptive advertising now implicitly to tell the smoker that he is in danger of heart disease or she chances a spontaneous abortion by smoking specific brands of cigarettes? This is a gamble that I do not think the Congress, or indeed the FTC, which has for several years advocated rotating explicit warnings, should take.

As an advertising man of 30 years' experience, I am worried, too, about another gamble involved in this bill. Very few advertising copy themes, illustrations or headlines nowadays get into public print or on the air without adequate research, without the principals being convinced that the message and all its connotations will be received in the manner in which the advertiser intended it. The testimony and questioning of witnesses here a week ago has revealed that there has been no research on the effect of the various warnings proposed in H.R. 5653.

Testimony before the full Commerce Committee in 1965 from psychology and marketing experts on the originally proposed warning label warned of the dangers of overstatement. One witness, the chairman of the department of marketing at Notre Dame, told the committee that to overstate the dangers of smoking could lead to an undermining of the effectiveness of warnings in use in connection with other products. "We all know the story of the boy who cried wolf," he said. "If warnings on cigarette packages portrayed smoking as a more serious or immediate danger than our experience confirms, we would soon come to attach little significance to the warnings."

A licensed psychologist and associate dean of the graduate school of business administration at Michigan State University went even further in his caution to the committee. Referring to the FTC's proposed warning requirement then in 1965, with its reference to "death from cancer and other diseases," he said, "In my judgment, a warning expressed in such language would, by virtue of its distastefulness and intended shock effect, precipitate a far greater backlash than a quieter, more restrained warning statement."

His fear was echoed in 1977 by psychologist Daniel Horn, the longtime head of the government's anti-smoking bureau, the National Clearinghouse for Smoking and Health, and a pioneer in education against smoking. Dr. Horn said, "I was always for the health warning [but] I was always concerned really about the level of strength in the health warning, partly because if a warning is too strong it becomes counterproductive." He added that he did not see the health warning as "an important source of education."

I should like to conclude with some comments from a book I wrote last year called "The Trouble With Advertising...," a book in which I presented an affirmation of advertising arrived at by means of a critical view from the inside. The portion I would like to tell you about is from a chapter entitled "What Advertising Isn't," in which I noted a 1979 FTC staff report, "Consumer Information Remedies." What Dr. Horn would call "education" is what the commission staff writers call "consumer information. These staffers, in discussing how to evaluate consumer information, wrote:

"The Task Force members struggled long and hard to come up with a universally satisfactory definition of the value of consumer information. Should the commission consider a mandatory disclosure to be a valuable piece of information, for instance, if it were later shown that although the consumers understood the information, they did not use it when making purchasing decisions? Is there a value in improving the quality of market decisions through the provision of relevant information, or is it necessary for the information to change behavior to have value?"

I wrote that "the ensuing 'remedies' make it clear that the staff really judges the value of mandatory disclaimer by the degree to which it changes consumer behavior in the direction they are seeking."

I pointed out that I'm a consumer and I resent government officials wondering what to do with me next if I understand but choose to ignore a disclaimer they've forced an advertiser to put in his ad? It's my God-given right to ignore any information any salesman presents me with -- and an ad, remember is a salesman. What's this about changing behavior? "Well, mine," I wrote, "is going to change if the employees of a government I'm paying for are talking like that out loud. It's going to get violent," I wrote.

These FTC persons -- indeed, with all due respect, Mr. Chairman, perhaps even you and the members of the subcommittee -- do not understand the nature of advertising, just as the professional critics of advertising, the journalists, consumerists, academicians, don't understand that advertising is not journalism or education and cannot be judged on the basis of objectivity and exhaustive, in-depth treatment.

Thorough knowledge of a subject cannot be derived from an advertisement but only from a synthesis of all relevant sources: the opinions of others, reports in newspapers, magazines and, increasingly, television -- even the advertising of competitors, which in the case of cigarette advertising gives the smoker the information he may seek in terms of tar and nicotine levels. I might add, too, that it is not just the warning on the pack and in advertising that has gotten across the message that the Congress decided upon, first in 1965 and then in 1969. It is the efforts of the American Cancer Society, the American Lung Association and the American Heart Association in spreading abroad the purported dangers of smoking.

Mr. Chairman, members of the subcommittee, I thank you for your time and patience. I hope you will remember when it comes time to vote upon H.R. 5653 that thorough knowledge of a subject cannot be derived from an advertisement and that by all reports, the American public is already thoroughly aware that smoking may be dangerous to health. Listing specific dangers of individual brands can only lead to overkill and discriminate against manufacturers of those brands which, in the roulette system to be devised by the Federal Trade Commission, may be forced to bear the label most explicit -- and most relevant to most smokers.

Statement by
L. G. S. Rao, Ph.D.
Bellshill Maternity Hospital
Bellshill, Scotland, U.K.
Regarding H.R. 4957

My name is Dr. L.G.S. Rao; I am Senior Biochemist at Bellshill Maternity Hospital in Glasgow, Scotland. I obtained my Ph.D. in Biochemistry from the University of Newcastle in 1966. I am the author of numerous scientific publications and have made presentations at scientific meetings in Europe and the United States.

My experience in clinical biochemistry over the past 20 years has been varied and has resulted in the development of interests in several methodological and clinical problems including perinatal medicine. Over the past several years, I have become deeply interested in the investigation of the causes of the high incidence of low birth weight and perinatal mortality which is found among the poorer patients of Bellshill Maternity Hospital. I have attempted to define in biochemical terms the risk factors associated with the "poor social conditions" which are supposed to be the cause of the poor reproductive performance of these mothers. I have found, as have other researchers in this field, that the biological characteristic of the poorer social groups that is of relevance to fetal growth retardation is their poor nutritional status and not, as has been claimed, their smoking habits. This finding could be of considerable practical importance because nutritional deficiencies can be corrected by dietary advice or supplementation. A program of such dietary intervention could lead to a striking reduction in the incidence of low birth weight and perinatal mortality and morbidity. Set forth below is a review of the scientific research supporting the above conclusion:

Introduction

There is a widely held view that maternal smoking is a cause of low birthweight (LBW) and perinatal mortality* (PNM) (the "causal" hypothesis). That view is challenged herein because the statistical association between smoking and LBW and PNM lacks the specificity and the consistency to be of causal significance and because of evidence inconsistent with the causal hypothesis.

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*The term perinatal mortality includes both stillbirths and infant death within the first few weeks after birth.

Most of the studies which favour the causal hypothesis have not corrected for factors which are already known to have a marked effect on LBW and PNM. The most conspicuous fact that emerges from a scrutiny of all the studies on smoking in pregnancy is that the so-called "effect" of smoking is seen only in the poorer underprivileged mothers and not in the mothers who have a good family income. Low family income could lead to nutritional deficiencies which can cause fetal growth retardation. Therefore, the so-called "effect" of smoking seen in only the poorer mothers may not be due to smoking itself, but due to deficiencies in maternal nutrition during pregnancy. Evidence for this has come from a recent study on the protein intake in pregnancy in Bellshill Maternity Hospital which showed that among mothers with normal protein intake, there was no difference in the proportion of LBW infants between smokers and non-smokers.

Other evidence against the causal hypothesis from a review of the available literature is also presented.

Smoking in pregnancy has become such an emotional issue that it is difficult to be unbiased in the design of the studies on this subject or in the interpretation of the results. As there is a great deal of evidence against the "causal" hypothesis, other hypotheses are required to explain the higher incidence of low birthweight infants and higher perinatal mortality found in some studies on smoking and pregnancy. There is considerable evidence for the view that fetal growth-retardation is mediated through a reduced weight gain in pregnancy, possibly due to undernutrition. This view will be denoted as the "nutritional" hypothesis. Another alternative hypothesis is the view that smoking is a characteristic of a certain type of person or a group of people who have a poor reproductive performance because of constitutional reasons. This hypothesis will be denoted as the "constitutional" hypothesis. The latter two hypotheses have several features in common and are complementary to each other. The evidence in favour of the "causal" hypothesis is presented in great detail in the 1979 U.S. Surgeon-General's report (1) and will not be discussed in detail, but the evidence in favour of the two alternative hypotheses will be presented and their relative merits discussed.

For a valid discussion of smoking and pregnancy, the effect of factors that are already known to affect birthweight and perinatal mortality needs a brief consideration.

Factors that are known to affect fetal growth:

Biological Factors

The biological factor which shows the best correlation with the weight of the infant is the functional capacity of the placenta, which is determined mainly by the size of the placenta and the quantity and quality of the blood supply to the fetus. The size of the placenta is largely determined by the height and weight of the mother. The factors that influence the quantity of blood flow are not well understood except in pathological states such as pre-eclampsia and extensive infarctions in the placenta. There is some evidence that uterine blood flow is under hormonal control (26). The quality of the blood supply is mainly determined by the nutritional status of the mother. It is well-known that in poorer countries with nutritional deficiencies, the weight of the mother and the infant are both lower than that of those in the more prosperous countries (2). Thus, maternal stature and the nutritional status during the pregnancy appear to be important factors on theoretical grounds and are, in fact, found to be so in practice. The other biological factors which are known to affect birthweight are:-

1. The length of gestation, the shorter the gestational period, the smaller the baby,
2. Sex of the infant, males being slightly heavier than females (about 200g) after about 36 weeks of gestation, and

3. The pregnancy number, the first child being slightly lighter than later children (about 200g).

Socio-economic factors

In addition to these biological factors, it is well known that there are important socio-economic factors which are known to influence birthweight. It is not surprising that economic status could have an influence on birthweight in poor countries, probably by affecting the nutrition of the mother, but even in some prosperous western countries the weight of the infant is related to the social and educational status of the husband. Thus, social classes 1 and 2 (professional workers) have a higher birthweight, longer gestational periods, lower perinatal mortality and lower congenital deformities than those in social classes 4 and 5 (manual and unskilled workers). These socio-economic factors appear to be particularly striking in their effect on perinatal mortality in the United Kingdom. Thus, the perinatal mortality in social class 1 was 9.5 per 1,000 births and was 32.0 per 1,000 in social class 5 (3).

The striking effect of social class on perinatal mortality appears to be mainly due to the higher incidence of prematurity, low birthweight babies, and congenital abnormalities. In fact, social class has such a striking effect on the health of the population in certain countries (4) that there is a higher incidence of shorter mothers (44%) in social classes 4 and 5 compared to social classes 1 and 2 (20.6%). The number of mothers delivered before 36 weeks gestation was twice as high for classes 4 and 5 (8.4%) as that of 1 and 2 (4.2%). (See Table 1). This higher incidence of prematurity alone can account for the higher perinatal problems of the social classes 4 and 5. The socio-economic differences persist even when maternal stature has been accounted for (5). It has been shown that the incidence of prematurity is associated with the social class of the mother's father, for any given social class of the mother's husband (6). This indicated that the socio-economic status (probably nutritional status) of the mother when she was a child had a striking effect on her growth in childhood and her reproductive performance in later life. Thus, in Britain, social class appears to affect the maternal size and nutrition, and could be a very important factor which affects birthweight and perinatal mortality. In the United States also, similar socio-economic factors appear to affect perinatal mortality. Thus, black populations in general and the less educated among the whites have a higher perinatal mortality than the better educated whites (29). In a large study on perinatal mortality conducted in Canada, the hospital status of the mother, whether private or ward patient, which is probably determined by the educational status of the father, has been shown to be an important risk factor, the private patients having a much lower perinatal mortality than that of the ward patients (21).

Thus, there are several important biological and social factors which affect birthweight and perinatal mortality and these must be taken into account when considering the association between smoking and perinatal mortality.

Some Methodological considerations

Most studies on the subject of smoking and pregnancy have not taken the above factors into consideration and their conclusions, therefore, are open to question. The 1979 Surgeon-General's report has pointed out that "problems arise from lack of adjustment for differences between smokers and non-smokers in the distribution of such factors as age, parity, socioeconomic status and race when the relationship of maternal smoking to perinatal mortality is under study." (1)

The usual definition of a low-birthweight baby is one that weighed less than 2500 grams at birth. Although it is a convenient definition, it could be subject to serious errors. The gestational age, the parity, and the sex of the infant must be taken into consideration to obtain some degree of validity of fetal growth retardation. It is obvious that for a small woman weighing 100 pounds, a 2500 gram baby born at 34 weeks is not a small baby but it is for a tall woman weighing 160 pounds and delivering at 40 weeks of her pregnancy. In fact, the 2500 gram limit appears to be absurd in some situations as it has been shown that the perinatal mortality for the under 2500 gram babies increases with the increase in maternal height (7). Although it would become too cumbersome to take the mother's height and weight into consideration in defining low birthweight, at least the gestational age, parity and sex of the infant should be taken into account.

An important concept in defining fetal growth retardation was described by Gruenwald (8) and adopted in a study by Miller and co-workers (9). According to this concept, fetal growth retardation is divided into two types. One is the "long, thin baby," which is the result of wasting that occurs during a period of days prior to birth, resulting in a low ponderal index. The second type has a general decrease in growth probably extending over a period of weeks before birth, with the result that the deficits in body length and weight at birth are proportional resulting in a normal ponderal index. The second type is described as short for dates (SHFD). The infants of the first type with the low ponderal index, have large appetites and will catch up within a few months with the weight of infants with a normal birthweight. On the other hand, the SHFD infants and their siblings have a notable incidence of organic disease, suggestive of a genetic or familial pattern. It can be seen later in this paper that this type of classification of fetal growth retardation is useful in the understanding of the question of smoking and pregnancy.

The "Causal" Hypothesis

Several studies have reported a higher incidence of low birthweight babies and a higher rate of perinatal mortality to be statistically associated with maternal smoking during pregnancy. However, there are some studies in which this association is not reported. Nevertheless, this statistical association has been interpreted as having causal significance, in spite of considerable evidence against this view. It may be worthwhile to assess critically the evidence for and against this hypothesis.

If maternal smoking is the cause of fetal growth retardation and an increase in perinatal mortality, this effect of smoking should be found in all countries and in all sections of the population. However, this is not found to be true. This effect is seen only in poorer social groups, but not in the upper socio-economic groups (See Table 2). Many studies have shown that the perinatal mortality is not significantly higher in the better-educated U.S. whites, or in the upper social classes in Britain (social classes 1 and 2). On the other hand, smoking is associated with higher perinatal mortality in only certain poorer groups such as the less educated whites and blacks in the U.S.A., and the manual and unskilled workers in Britain. This lack of consistency of the so-called "effect" of smoking is against the causal hypothesis.

Fetal growth retardation is not specific to the babies of mothers who smoke in pregnancy. Other associations such as maternal stature, maternal nutrition and socio-economic status are already shown to be of causal significance.

The strength of the reported statistical association between maternal smoking and perinatal mortality is much less than that of other factors such as previous history of perinatal loss or socio-economic status. For example social class 5 (unskilled workers) in Britain shows an excess of perinatal mortality of 400% over that of social class 1. This excess is more than ten times that which is associated with smoking (about 35%). Such a comparatively weak statistical association is not in favour of the causal hypothesis.

The claimed dose-response relationship is often quoted as evidence for the causal hypothesis. In such studies, it is obviously important to keep other factors constant, while the different doses and their responses are compared. This is difficult except by statistical methods such as by the analysis of co-variance. Most of the studies which have reported a dose response relationship have not corrected for the influence of other factors. However, a study by Mayer and associates (21) have done this and claimed a small "independent" effect of smoking, with public hospital status and previous pregnancy loss showing much stronger associations with perinatal mortality. In a study of a large number of subjects (some 50,000) by Naeye (22) where all the factors that are known to affect birthweight were taken into account, the difference in the mean birthweight of babies of light smokers and of heavy smokers at 37-41 weeks of gestation was not significantly different. At 39 and 40 weeks of gestation with 8454 and 10300 subjects, at each week the difference in the mean birthweight was only 2 grams and 1 gram, respectively (See Figure 1). The results of this well controlled study using large numbers of subjects should be very reliable, but do not indicate a dose-response relationship.

The temporal relationship between the change in the incidence of disease and the amount of smoking in the population has been cited as evidence in the controversy regarding smoking and certain diseases e.g., lung cancer. In the case of smoking and perinatal mortality, there is an inverse temporal relationship. Smoking has markedly increased in the last thirty years in women, whereas perinatal mortality has markedly decreased during this period in most Western countries.

In view of the foregoing, it is necessary to consider some alternative hypotheses to explain the higher incidence of fetal growth retardation and perinatal mortality reported in some groups of mothers who smoke during pregnancy.

"Nutritional" hypothesis and the role of weight gain in pregnancy.

It is well known that in poorer countries with nutritional deficiencies, the weight of the mother as well as that of the baby are lower than that of those in prosperous countries. Nutritional supplementation is known to increase the average birthweight in these poor countries. It is generally assumed that in prosperous western countries, such as the U.S.A. and Britain, there are no nutritional deficiencies. However, several studies have shown that it is not true for all sections of the population in these countries. Proteins, vitamins and trace elements which are essential for the normal development of the fetus are the most expensive of foods and could be deficient in the diet of the mothers from low-income groups (11). In a study in the U.S.A., it has been shown that mothers from a low-income group ate less protein than mothers from a high income group (12). In another study in the U.S.A. nearly a third of the mothers (53 out of 182) had a low protein-low calorie intake and had a significantly higher incidence of low birthweight babies (13). Deficiency of an extremely important trace metal, namely zinc, can occur in certain sections of the population in the U.S.A. (14). The possible importance of undernutrition as a cause of fetal growth retardation is reflected in the higher incidence of low birthweight babies in the poorer sections of the population in the U.S.A. and the U.K. For example, the upper social groups (social classes 1 and 2) have an incidence of low birthweight of 4.5% whereas for the lower social classes it is 8.2% (15). The perinatal mortality per 1000 births (3) for the upper social classes is 9.5 whereas for the lower social classes (social class 4 and 5) it is 35. In the U.S.A., the incidence of low birthweight and perinatal mortality is higher for the babies of black mothers and the less-educated whites than it is for the babies of the better-educated whites. An extremely important fact that emerges from many of the studies on smoking and pregnancy is that the excess of perinatal mortality found in mothers who smoke is not found in the prosperous groups, but only in the poorer sections of the community in the U.S.A. and the U.K.

(See Table 2). It can be seen that perinatal mortality ratios of smokers' babies to those of non-smokers' babies, varies between 1.02 to 1.13 in seven studies in which the mothers were from the upper social groups whereas for the lower social groups the mortality ratio varied from 1.39 to 2.15. These results make it abundantly clear that the socio-economic status is very important in determining the incidence of perinatal mortality.

A large number of studies have reported a higher incidence of low birthweight babies for mothers who smoke during pregnancy since Simpson in 1957 first made this observation (See ref. 1 for further references). As mentioned before, most of these have not taken into account factors which affect birthweight such as social class. In a few studies which have taken these factors into account there is no significant difference in birthweight between smokers' and non-smokers' babies (See Table 3).

The possible role of nutrition is highlighted in a recent study conducted on mothers from Bellshill Maternity Hospital (16). The birthweight was adjusted for the gestational age, parity of the mother and the sex of the infant (4). The mothers were classified according to the social class of the husband. It was found that the mean birthweight of the upper social classes 1, 2 and also 3 (professional and technical) did not show a significant difference between smokers and non-smokers whereas this difference was large and highly significant in the lower social classes namely 4 and 5 (manual and unskilled.) The incidence of small-for-gestational-age ("SFGA") infants was significantly different between smokers and non-smokers in only the lower social classes and was greater in social class 5 than in social class 4. (See Table 4). If smoking during pregnancy is the cause of fetal growth retardation, why does it occur only in certain groups of mothers and not in others? It can scarcely be argued that the so-called "effects" of smoking respect the upper social classes and pick on only the poor and underprivileged mothers.

The results from this study give an excellent example of how the same data can be interpreted in different ways, either for or against the "causal" hypothesis. The incidence of SFGA infants for all social classes as a whole was 6.4% for non-smokers and 15.2% for smokers. This difference in incidence of SFGA infants between smokers and non-smokers is statistically significant ($p < 0.01$). It would be quite easy to claim from these results that smoking was the cause of the higher incidence of SFGA infants in smokers, as has been done in many other studies. It would also be quite difficult to disprove this claim with this experimental design. However, if an attempt is made to make smoking the only variable between smokers and non-smokers by correcting, as far as possible, for factors that are known to affect birthweight, the results are no longer in favour of the causal hypothesis, and are in fact against the causal hypothesis. Thus, if smokers were classified according to the social class of the mothers' husband, entirely different incidences of low birthweight are seen in the different social classes for the same amount of smoking, and smoking mothers from the upper socio-economic groups do not show an excess of incidence of SFGA infants. If smoking does not cause fetal growth retardation in one social group, it cannot obviously be the cause in another ethnically same social group. Therefore, the association between the higher incidence of SFGA infants and smoking in the poorer social groups cannot be considered to have a causal relationship. A causal relationship might be claimed if smoking were the only variable between smokers and non-smokers. Most studies on smoking and pregnancy assume that the smoking habit is the only difference between the life-style of smokers and non-smokers.

Contrary to this assumption, there is much evidence that smokers and non-smokers differ in their personality, their attitude to life and their eating and drinking habits (23). A significant difference in the drinking habits alone could account for the higher incidence of SFGA infants in certain groups of smoking mothers. Therefore, it is entirely unjustified to claim that smoking was the cause of fetal growth retardation in any group of smokers, unless the smoking and non-smoking mothers are exactly comparable in every other way.

The gradient in the incidence of SFGA infants with decrease in socio-economic status (Fig. 4) of smoking mothers indicates that some factor such as family income, which is quantitatively associated with the social class gradient, is also quantitatively related and possibly bears a causal relationship to fetal growth retardation. Since family income could affect the nutritional status of the mother, maternal nutrition may be the cause of the higher incidence of fetal growth retardation in the poorer social groups. This view has the advantage that it will lend itself to experimental verification and has been recently verified on a group of mothers in Bellshill Maternity Hospital.

Maternal nutritional status was investigated by measuring the protein intake in the third trimester of pregnancy. Those mothers who had a low protein intake in pregnancy had a disproportionately higher incidence of SFGA infants (43%) compared to those with a normal protein intake (3.4%). See Table 6. Among mothers who smoked, those who had a normal protein intake had the same low incidence of SFGA infants as non-smokers. This indicates that nutritional deficiency, but not smoking, is a pre-requisite for the development of fetal growth retardation. Since the majority of mothers who were delivered of SFGA infants (65 out of 95, 68%) had a low protein intake, correction of this and other associated nutritional deficiencies is likely to result in a striking reduction in the incidence of low birthweight, irrespective of their smoking habits.

Further evidence in favour of the nutritional hypothesis comes from studies on maternal weight gain during pregnancy. It has been suggested that smoking mediated its so-called "effect" through maternal weight gain; Rush (17) and Davies and associates (18) have shown that the mean weekly weight gains were reflected in the infant's birthweight. Davies et al. reported that their results are consistent with the view that mothers' own weight gain has a greater effect on fetal growth with smoking having a very small "independent" effect.

Other studies (19, 20) investigating the role of weight gain during pregnancy have reported an independent "effect" of smoking when weight gain was held constant. However, when an analysis of variance was carried out (21), previous pregnancy loss and hospital status (private or public) showed a much higher degree of statistical correlation (F. ratio 97.4 and 44.2 respectively) than smoking (8.4). Miller and associates (9) have shed some light on the role of weight gain in fetal growth retardation. They found in a study of 688 non-smokers and 424 smokers that there were 62 and 67 mothers who gained less than 0.5 pound per week in the last two trimesters of pregnancy. The incidence of fetal growth retardation was five times higher among non-smoking mothers with low weight gain (less than 0.5 pound per week) than among mothers with greater weight gain. For smokers, it was three times as high among those with low weight gain than among those with greater weight gain. In the group of 42 mothers who delivered infants with fetal growth retardation, the incidence of mothers who were in the lower social classes and who smoked were significantly higher than the respective incidences among 1070 mothers who did not have infants with fetal growth retardation.

Further evidence for the importance of weight gain in pregnancy is provided by the work of Naeye (22). He studied the weight gain in successive pregnancies in groups of smokers, non-smokers and two groups of women who smoked only in either the first or the second pregnancy. Naeye found that mothers who smoked during one pregnancy but not in another had smaller infants in the pregnancy in which they smoked irrespective of the birth order, and a variety of other maternal and fetal factors that are known to affect fetal growth. He claimed, therefore, that smoking appears to have an independent effect on fetal growth. However, close examination of his data (Table 3 of his paper) shows some interesting correlations between maternal weight gain and birthweight. Since this author has recorded maternal weight gain and birthweight in both the first and second pregnancies in which smoking was the only variable, it is possible to compare the difference in maternal weight gain with the difference in birthweight between the first and second pregnancies. Such a comparison showed that the differences in weight gain between the first and second pregnancy had a highly significant correlation with the difference in birthweight between the successive pregnancies (See Figure 2). This clearly indicates that weight gain is the factor that determines birthweight, and smoking, contrary to his claims, does not have an independent effect. Since maternal nutrition has probably the most important effect on the weight gain, these results can be considered as strong evidence in favour of the nutritional hypothesis.

Constitutional Hypothesis: Smoker or Smoking?

Whether low birthweight and higher perinatal mortality is due to smoking or due to the smoker (smokers' biological and social characteristics) is a real and important question. Abundant evidence is presented above to show that smoking has no statistically significant association with low birthweight or perinatal mortality in the upper social groups, but only in the poorer, underprivileged and probably undernourished groups of mothers. In addition to these socio-economic characteristics, there are probably also genetic, biological and psychological characteristics of some smokers who will have a poor reproductive performance. It has been shown repeatedly that the poorest of the social groups have also the largest proportion of smokers. It is reported that the eating and drinking habits of the smokers are very different from those of non-smokers (23). Yerushalmy has (23) produced some good evidence in favour of the "constitutional" hypothesis. He showed that the proportion of low birthweight babies born to mothers who became smokers in later pregnancies was significantly higher than those of mothers who never smoked, and was similar to that of the babies of mothers who smoked in all their pregnancies, indicating that certain types of mothers have low birthweight babies, whether they smoked or not. In commenting upon these findings, the 1979 U.S. Surgeon-General's report (1) claimed that these results are not reliable because the mean age of the "future" smokers was 19.7 years and was significantly different from that of the non-smokers which was 22.1 years. Although Yerushalmy during his lifetime, had adequately replied to this type of criticism (24), his reply is not taken into account by the 1979 U.S. Surgeon-General's report and may therefore be worthwhile reiterating. Figure 3 drawn from Yerushalmy's data (24) is a regression of maternal age and the incidence of the percentage of low birthweight babies. It can be seen from the regression line the percentage of such babies at 19.7 years is 6.44% and for 22.1 years, it is 5.89%, a difference of 0.55% which is not significant by the Chi-Square test. On the other hand, the difference between the percentage of low birthweight babies of future smokers (9.5%) and non-smokers (5.3%) is significant at $p < 0.02$. It is also of interest that mothers who gave up smoking in later pregnancies had significantly less low birthweight babies before they gave up smoking than those who smoked in all their pregnancies. (23) Silverman (25) set out to confirm these findings, but her results were equivocal and could not either confirm or deny the findings of Yerushalmy. Further work along these lines is required to answer the question whether it is the smoking or the smoker that is the cause of low birthweight and higher perinatal mortality reported in some groups of smokers.

It should be pointed out that the higher incidence of low birthweight babies does not necessarily result in increased perinatal mortality. Table 5 shows that the perinatal mortality rate of such babies of smoking mothers is actually lower than those of non-smoking mothers. This paradoxical finding has never been adequately explained by the "causal" hypothesis.

Gruenwald's and Miller and associates' classification of fetal growth retardation into those with normal length for gestation with a low ponderal index (PI) and those with retardation in length and weight (SHFD) is also of relevance to the discussion of the constitutional hypothesis. The proportion of babies with a low PI was greater among smokers than among non-smokers (2.3% in non-smokers and 3.0% in smokers). If smoking causes hypoxia to the fetus and other ill effects one would not expect a higher proportion of low PI babies among smokers but just the opposite. The other category of fetal growth retardation, namely, short for gestational age (SHFD), is of much more serious pathology, suggestive of a genetic or familial pattern. Babies of smoking mothers have an excess of the latter type of growth retardation, indicating that this disorder was genetic or constitutional. As mentioned before, the mothers of such infants had a disproportionate number of women from poor social class. Furthermore, the placental weight of the mothers who had SHFD infants was significantly lower than that of normal, whereas in conditions which result in hypoxia such as altitude, placental weights are larger than normal. Under these conditions, it would be unjustified to assume that either type of fetal growth retardation was caused by smoking.

We are still far from understanding the etiology of fetal growth retardation. We do not fully understand the normal physiology of pregnancy. However, there are certain clues to the etiology of fetal growth retardation. The factors that are known to have an effect on fetal growth have been mentioned before. The quantity of blood supply to the fetus is obviously an important factor, and this appears to be affected by hormonal influences. Unconjugated free oestriol and oestradiol appear to affect uterine perfusion (26) and an increase in blood volume which occurs in pregnancy. It has been shown that maternal intravascular blood volume in pregnancy is highly correlated with fetal weight (27). As the precursors for these hormones are of fetal origin, the fetus may control its own growth potential to a large extent. Thus, the vitality of the fetus which is of genetic origin probably has the most important influence on its own growth with effects of maternal and evironmental factors superimposed upon it.

In conclusion, the evidence against the "causal" hypothesis is so considerable that alternative hypotheses are required to explain the findings of studies on smoking and pregnancy. On the other hand, the evidence for the "nutritional" and "constitutional" hypothesis is far more convincing. It is important to remain unbiased and be objective in designing studies on smoking during pregnancy and in the interpretation of their results. All the available evidence suggests that the higher incidence of fetal growth retardation in certain groups of mothers who already have a poor reproductive performance is a social problem for which smoking is not the cause but only a symptom. It is also obvious that the solution of this serious social problem, i.e. lack of proper nutrition, will be neglected if undue attention is given to smoking.

Table 1.

Incidence of Certain Characteristics in Primiparae by Husband's Social Class.

Thomson et al 1968.(4)

Husband's Social Class	Delivered at 36 weeks or earlier (per cent)	Women below 62 inches (157.5 cm.) (per cent)
I and II	4.2	20.6
IIIa	6.5	32.9
IIIb	6.5	38.1
IV and V	8.4	44.0

Table 2.

PERINATAL MORTALITY IN RELATION TO SMOKING.

	Non-Smokers number of subjects	Smokers number of subjects	Perinatal loss/1000 Non-Smokers	Perinatal loss/1000 Smokers	Ratio	Reference
1. British Perinatal Mortality Survey Social Class 1 and 2 Butler et al. (1968)	11,145	4660	25.8	26.3	1.02	28
2. Boston City Hospital } White Rush and Kass (1972) }	513	892	29.2	31.4	1.08	29
3. U.S. Collaborative Perinatal Survey Niswander and Gordon (1972) White	8,521	11.369	31.4	35.5	1.13	30
4. Comstock et al. (1971) (Fathers education 9+ years)	7.646	4,641	29.2	31.4	1.12	31
5. Underwood et al. (1969)	24,865	23,629	19.7	20.8	1.05	32
6. Rantakallio (1969) (Northern Finland)	8,898	2,368	23.2	23.4	1.08	33
7. Fabia (1977) (Canada) (Maternal age 25-34)	3,192	2,962	12.6	13.2	1.05	34

Table 3.(Contd)

STUDIES WHICH HAVE SHOWN A HIGHER PERINATAL MORTALITY IN SMOKERS

		Perinatal loss/1000			
		Non-Smokers	Smokers	Ratio	Reference
1. British Perinatal Mortality Survey Social Classes 3, 4 and 5 Butler et al. (1968)		33.5	46.6	1.39	28
2. Rush and Kass (1972)	Black	28.6	54.1	1.89	29
3. Comstock et al. (1971)	White <8 years education	17.6	38.0	2.16	31
4. Fabia (1977)	White >35 years	23.6	41.7	1.81	34

TABLE 4. BIRTHWEIGHT ACCORDING TO SOCIAL CLASS AND SMOKING

(From Rao, 1979, ref. 16)

Social Class	NON-SMOKERS				SMOKERS			
	n	Birthweight g ± 1SD	S.F.D. n	S.F.D. %	n	Birthweight g ± 1SD	S.F.D. n	S.F.D. %
1.	74	3504 ± 450	4	5.40	25	3398 ± 416	–	0
2.	226	3471 ± 502	13	5.75	74	3352 ± 564	5	6.75
3.	496	3375 ± 528	32	6.45	233	3259 ± 527	18	7.27
4.	657	3452 ± 512	42	8.46	626	3178 ± 496*	95	15.17*
5.	168	3396 ± 548	14	7.14	271	3101 ± 566*	69	25.46*

* Significant difference between smokers and non-smokers $p < 0.001$

S.F.D. = Small for the gestational age, parity and sex of infant.

TABLE 15

Perinatal deaths among all infants and among low-birthweight infants by mother's smoking status in four large studies

Investigator	Year	No. of Births	Per cent L.B.W. Infants		Perinatal deaths/1000			
					All Infants		LBW Infants	
			Smoker	Non-smoker	Smoker	Non Smoker	Smoker	Non Smoker
Underwood & Associates (34)	1967	48505	8.9	5.7	20.5	19.7	187.5	269.0
Rantakallio (34)	1969	11931	6.1	3.5	23.4	23.2	287.5	343.6
Butler & Colleagues (29)	1969	16954	9.3	5.4	44.8	32.4	268.5	284.5
Yerushalmy* (28)	1971							
White		9793	6.4	3.2	11.3	11.0	113.9	218.3
Black		3290	12.3	5.8	21.5	17.1	113.8	201.6

* Neonatal deaths only

Table IX Incidence of Low birthweight (LBW) according to protein intake in smokers and non-smokers

	Non-smokers	Light smokers	Medium smokers	Heavy smokers	Total
Number of pregnancies	470	189	224	127	1010
LBW in normal protein intake	16 out of 422 3.78 %	4 out of 173 2.3 %	6 out of 175 3.4 %	4 out of 95 4.2 %	30 out of 865 3.4 %
LBW in low protein intake	12 out of 47 25.5 %	9 out of 18 50 %	36 out of 48 54 %	18 out of 32 56.2 %	65 out of 145 44.8 %

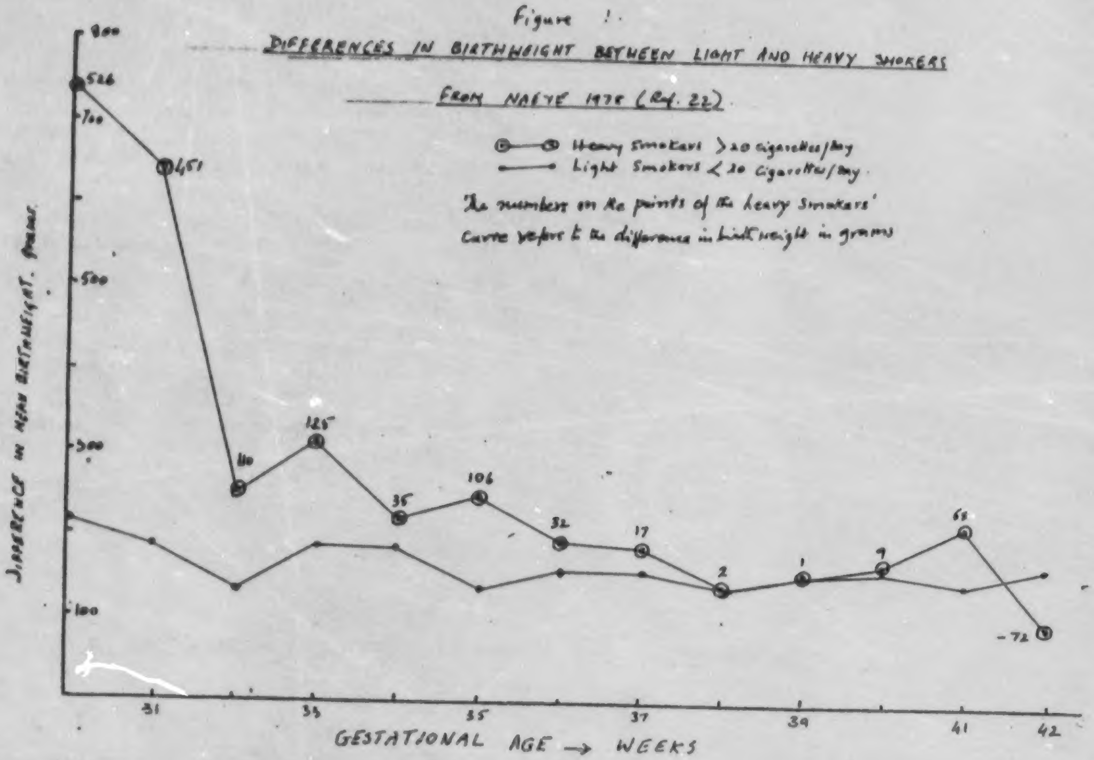
LBW in all smokers:	Normal protein:	14 out of 540	= 3.81 %
	Low protein:	53 out of 98	= 54 %
All LBW infants with low protein:		65 out of 95	= 68 %

Light smokers (less than 10 cigarettes a day); Medium smokers (10-19 cigarettes a day)
Heavy smokers (20 or more cigarettes a day)

Figure 1.

DIFFERENCES IN BIRTHWEIGHT BETWEEN LIGHT AND HEAVY SMOKERS

FROM NAEYE 1978 (Ref. 22)



The numbers on the points of the heavy smokers' curve refer to the difference in birth weight in grams

FIGURE 2

CORRELATION OF DIFFERENCES IN WEIGHT GAIN AND BIRTHWEIGHT BETWEEN FIRST AND SECOND PREGNANCIES

Based on data from Table 3 of Waaye's Paper.

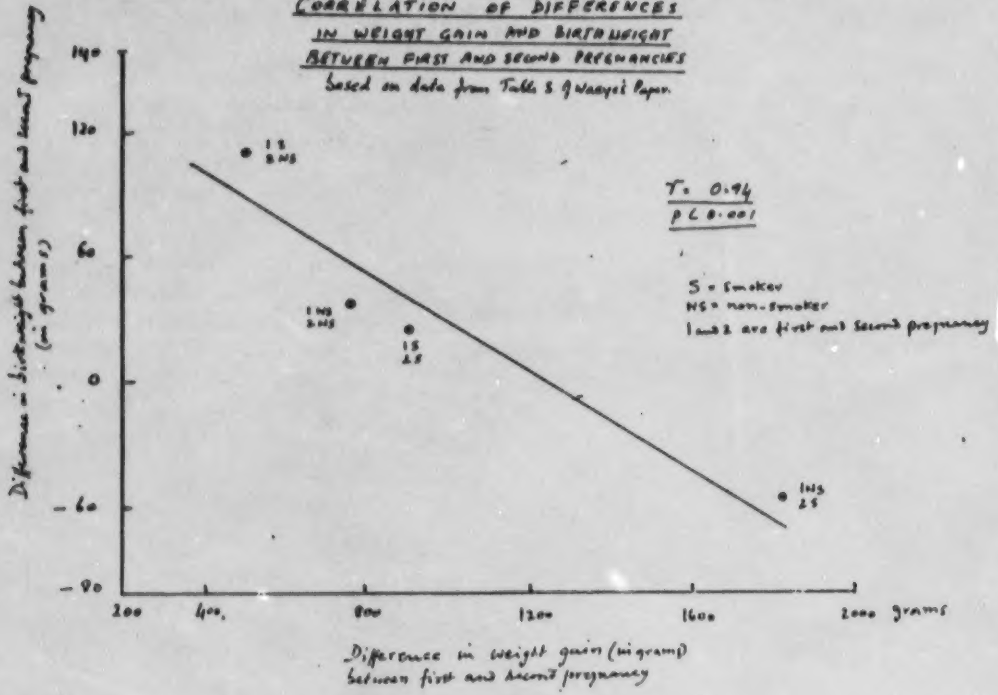
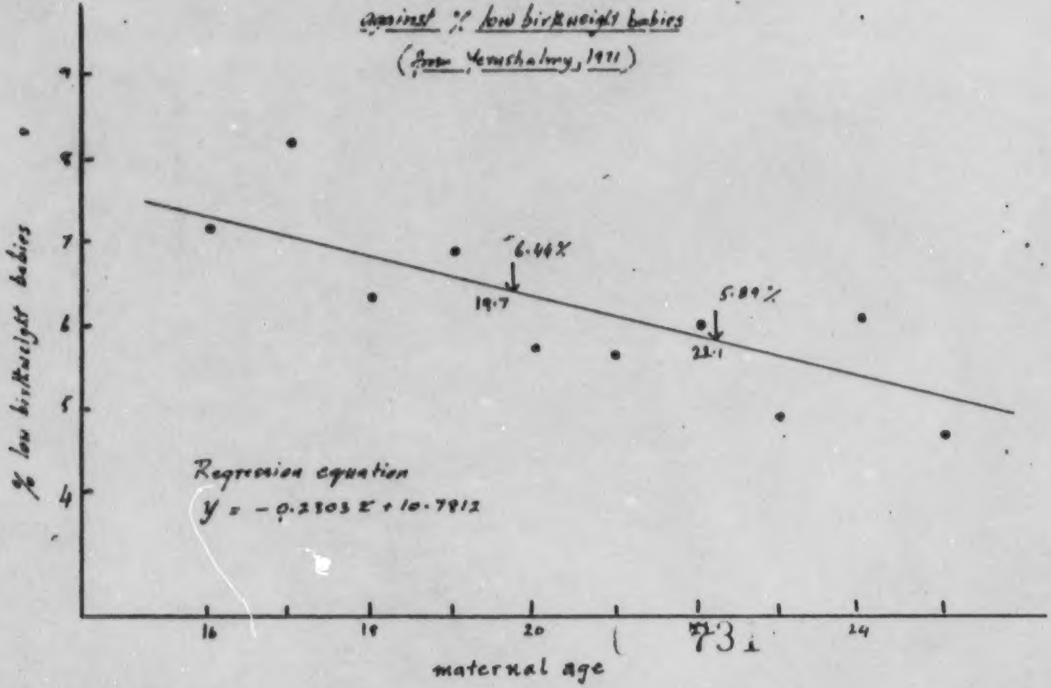


Figure 3

Regression of maternal age against % low birthweight babies

(from Yerushalmy, 1971)



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STATEMENT OF DR. JAY ROBERTS

I am Dr. Jay Roberts, Professor and Chairman of the Department of Pharmacology at the Medical College of Pennsylvania in Philadelphia. I also have served as a consultant for the National Research Council, NIH study section groups, and a number of private companies and laboratories. I have also been a Research Associate on the consulting staff at the Philadelphia Geriatric Center.

My current activities in the scientific research field include direct laboratory experimentation and editorial and reviewing responsibilities for scientific publications. I am on the Editorial Board for the Journal of Clinical Pharmacology, Journal of Cardiovascular Pharmacology and Journal of Gerontology.

The professional societies to which I belong include the American Society of Pharmacology and Experimental Therapeutics, the Society for Experimental Biology and Medicine and the American College of Clinical Pharmacology. I am a Fellow in the American College of Cardiology , the College of Physicians of Philadelphia and the Gerontological Society of America.

I have been asked to comment as a scientist on two bills pending before Congress, HR 4957 and S 1929. My comments will be limited to the scientific points that are raised in the proposed legislation.

My primary concern with both proposed bills is the conclusive nature of Section 2. A number of unanswered, scientific questions come to mind:

1. To what extent has the physiological and pathological changes associated with aging been taken into account?

2. Where are the data that conclusively prove one-third of heart disease deaths are attributable to smoking?

3. How much and to what degree do occupational exposures contribute to chronic disease statistics?

4. What is the relationship between smoking, aging and the physiological and psychological constitution of the individual? How do these relationships affect disease rates?

While statistical correlations are important and suggestive,the scientific conclusions reached need to be explored further. Smoking per se is not the only factor or characteristic of smokers that needs to be examined in studies. For example, the effect of stress on the cardiovascular system is well known and exposure to high levels of stress for long periods of time may have a major impact on the health of certain people. Another complicating unknown is the relationship between stress and smoking; why do people smoke and what does it say about their attempt to deal with stress?

Our animal studies involving nicotine show that it produces acute cardiovascular effects. But we need to know more about the chronic effects of nicotine in animals. Similarly, we need to know more about how nicotine is handled in the human system and how this directly relates to changes in the cardiovascular system.

My experimental work both in the cardiovascular system and in aging areas leads me to conclude that the factors involved in the development of chronic disease are complex. Despite extensive research in animals and observational work in humans, there are many unanswered questions why and how people develop diseases of the cardiovascular system. Statements in the present bills, such as, "1/3 of the deaths attributable to heart disease are associated with smoking" would seem to imply that the answers to the questions I raise are already answered and no further scientific study of this matter is warranted. But one may ask whether the diseases of the cardiovascular system in question are a natural result of the aging process or do other factors such as genetic background and lifestyle habits initiate and promote the disease?

As a scientist, I am interested in research and its role in providing reliable scientific information. I believe through further research the questions raised above could and should be answered.

Statement of Henry Rothschild, M.D., Ph.D.

Introduction

I am Henry Rothschild, a medical doctor and doctor of philosophy specializing in genetics, molecular biology and oncology, currently Professor of Medicine and Anatomy at the School of Medicine, Louisiana State University, New Orleans, Louisiana.

Since 1962 I have been engaged in .research involving the genetic basis of disease and am director of the Louisiana Ethnogenetic Disease Association.

I currently serve as consultant to the Louisiana Department of Health and Human Resources and am a member of the Research Committee of the American Heart Association of Louisiana. I have published over 40 scientific articles. My Curriculum Vitae and list of publications are attached.

Discussion

Studies conducted in Louisiana have demonstrated an unusual distribution of lung cancer mortality. The lung cancer mortality in white males in Louisiana is the highest in the nation, and in particular the 27 southern preponderantly rural parishes ranked in the top 3% nationally from 1950 through 1976 and has risen since then. Studies that I have been conducting since 1975 on this population indicate that genetic factors may play a significant role in this excess mortality from lung cancer.

In our initial investigation we interviewed the next-of-kin of 284 of the 815 persons who died of lung cancer during 1971 through 1977 in the 10 southern, nonurban parishes. We found that 108 (38%) of the decedents had been employed for at least six months as sugarcane-farm workers at some time during their lives and that the relative risk estimate of lung cancer mortality for sugarcane-farm workers was more than twice that of the controls. Neither employment in other industries nor tobacco consumption could account for the elevated risk of lung cancer mortality associated with sugarcane farming.

We then began to explore the thesis expressed by many investigators including a group from the National Cancer Institute (Goffman, et al.,, 1982) that there may be "an inherited predisposition to lung cancer." Studies have shown that there are genetic factors associated with lung cancer pathogenesis. It has been found, for example, that first-degree relatives of individuals diagnosed as having lung cancer have higher rates of lung cancer that cannot be accounted for by other factors such as age, sex, birth cohort, or cigarette smoking. It was also shown that a non-smoking first-degree relative of an individual with lung cancer has about a fourfold risk of dying of lung cancer when compared to control relatives. (Tokuhata and Lillienfield, 1963, Tokuhata, 1964) A similar study reported an increased risk of lung cancer mortality in siblings of individuals with lung cancer (Fraumeni, et al., 1975)

The argument for the potential genetic regulation of tumor incidence (Bodeau et al., 1974) is supported by results of studies indicating that many mouse strains differ in their incidences for specific tumor types (Murphy, 1966). In humans, a spectrum of different genetic predispositions for each tumor implies that a distribution curve of genetic susceptibilities exists for each tumor (Reif, 1981).

Studies of distribution of cancer among different ethnic groups further support this proposition. Compared with whites, for example, blacks have a more rapidly accelerating incidence of lung cancer in the United States, Americans of Mexican and Chinese extraction (Buell et al., 1968; Fraumeni and Mason, 1974) also have a higher incidence, and American Indians (Creagan and Fraumeni, 1972) a lower incidence. Chinese in Hong Kong have an unusually high prevalence of adenocarcinoma of the lung, a relatively infrequent cell type in other areas (Belamaric, 1969).

In analyzying our data we obtained results that establish a strong genetic component for lung cancer in the Louisiana population. (Ooi et al., 1981). Of the

446 white residents of the target parishes who died of lung cancer during 1976-1979, we interviewed the next-of-kin of 328 cases. Of the 328 case families, 54 (16.5%) had first-degree relatives with lung cancer, compared with 16 (5.3%) in 304 control families. There were 61 (2.2%) lung cancers among the 2767 case first-degree relatives, compared with 18 (0.8%) in 2389 control relatives. This aggregation of lung cancer could not be attributed to differences in age at death, family size, or mortality. Six (1.8%) of the case families had two or more first-degree relatives with lung cancer. First-degree relatives of persons with lung cancer had, therefore, a lung cancer risk more than twice that of control relatives.

Comparing case and control relatives by stepwise logistic regression indicated that a person's relationship to the person with lung cancer, and the square of a person's age, are the two strongest predictors of lung cancer outcome, even after controlling for the effects of age and sex.

We are in the process of further investigating this population to attempt to ascertain whether any major genes can be isolated as being involved in the pathogenesis of lung cancer. If we can isolate such genes, it will be major step forward in unravelling the mystery of lung cancer causation.

Henry Rothschild

Henry Rothschild, M.D., Ph.D.

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STATEMENT OF DR. HENRY I. RUSSEK

I am Dr. Henry I. Russek, a practising specialist in the field of cardiovascular disease. I received my medical degree from the Royal College of Physicians & Surgeons in Edinburgh, Scotland. Formerly, I was Director of Cardiovascular Research at the U.S. Public Health Service Hospital, Staten Island, and Research and Clinical Professor in Cardiovascular Disease at New York Medical College. In addition, I served as consultant in cardiology at several other New York area hospitals.

I am a Fellow of the American College of Physicians, the American College of Cardiology, the Council on Clinical Cardiology of the American Heart Association, the American College of Chest Physicians and the International College of Angiology.

I am certified by the American Board of Internal Medicine and hold certification in the subspecialty of cardiovascular disease. I have published over one hundred and fifty scientific articles and have edited seven books concerned with diseases of the heart. Since 1968, I have been Program Chairman for the Annual Cardiovascular Graduate Symposium for the American College of Cardiology.

I have been asked to comment on proposed legislation in the current Congress (H.R. 4957 and S. 1929) which contains several statements describing cigarette smoking as a "cause" of heart disease. I would like to direct my comments to those conclusions.

For more than 30 years I have had a sustained interest in identifying the factors responsible for the high incidence of coronary heart disease among persons living in the United States. My early investigations conducted in young coronary patients and healthy controls clearly indicated that emotional stress of occupational origin was far more important statistically than high fat diet, obesity, lack of exercise or cigarette smoking in relation to prevalence of the disease.

Those early observations led to our investigation of the occurrence of coronary heart disease in professional groups in which there were significant differences in the demands and responsibilities of routine employment. In a questionnaire survey which we conducted among 25,000 persons in 20 occupational categories, a striking correlation was found between the prejudged stressfulness of occupation and the reported prevalence of coronary heart disease. Of further interest was the observation that the frequency and intensity of the smoking habit in different professional groups was directly correlated with the relative stressfulness of the occupational activity. Due to such confounding, we could not say whether smoking is independently associated with heart disease or whether it is simply related to what may be the real culprit, emotional stress.

My studies of emotional stress and clinical experience have led me to question the widely accepted view that high cholesterol, elevated blood pressure and cigarette smoking are the most important factors in the etiology of coronary heart disease. The fact is that these traditional risk factors are often absent in new cases of heart disease encountered in clinical practice. Moreover, I believe one can question seriously whether preventive measures directed against these "etiologic" influences have been successful. In this regard, our analysis of data on American physicians over a twenty-year period showed that there had been no significant change either in overall longevity or in average age at death from coronary heart disease. This observation made it evident to us that other risk factors must be involved because U.S. physicians are reported to smoke very little and no segment of our society is more aware of the potential dangers of hypercholesterolemia and hypertension.

Further insight into the etiology of heart disease was obtained during our analysis of data from large population surveys. We observed that persons who had stopped smoking of their own free will actually had a lower prevalence of coronary heart disease than persons who had never smoked. The original Framingham studies showed similar findings but no attempt was made to interpret this anomaly. Our explanation is that persons who discontinue smoking for reasons unrelated to medical necessity, fear or coercion, may possess an unusual capacity for adaptation to stress and thus a diminished vulnerability to atherogenic influences.

My clinical observations and research have led me to conclude that while smoking of cigarettes is harmful to the cardiac patient, many of the conclusions and findings about heart disease etiology need to be reexamined in a broader framework. Consistent and persuasive findings are being reported linking coronary heart disease to prolonged emotional stress, coronary-prone behavior patterns, sociocultural mobility, and stressful life events. Obviously, if cigarette smoking is involved in the pathogenesis of coronary artery disease, the relationship is far from clear and further research is needed in this complex area.

Henry I. Russek

Henry I. Russek, M.D.

Statement of Bernice C. Sachs, M.D.; Seattle, Washington

After graduating from the University of Michigan Medical School "with distinction" in 1942, I took my internship and post-graduate training at Michael Reese Hospital and Medical Center, and the Institute for Pyschosomatic and Psychiatric Research and Training, in Chicago, 1942-1949. Since that time, I have practiced psychiatry and psychosomatic medicine in a pre-paid Cooperative Plan which delivers comprehensive medical care to Seattle, Washington, and its environs. The Cooperative has 325 board-certified or board-eligible physicians who serve a population of 285,000, and it owns and operates two hospitals comprising 450 beds. It is one of the first, and one of the largest, Health Maintenance Organizations (HMO) in the United States.

I am President of the Academy of Psychosomatic Medicine, Trustee of the King County Medical Society assigned to the Drug Abuse and Alcoholism Committee, member of The Cooperative's Cancer Committee, Board Member of the Education and Research Foundation of the American Society of Clinical Hypnosis, and Chairman of its Research and Grants Committee. I am past Chief of The Cooperative's Mental Health Service (1970-72, 1976-78), past President of the American Medical Women's Association, American Society of Clinical Hypnosis, American Psychiatric Association's Seattle Branch, and past

Chairman of the Cooperative's Alcohol and Drug Abuse Committee. In my medical practice, I have seen thousands of patients and given talks to and met with thousands of persons who shared my interests in community health and welfare. My curriculum vitae and bibliography are attached.

The large body of psychiatric experience demonstrates that disease is a problem of the individual as a whole, and not an autonomous disturbance localized in a particular organ. The extraordinary complexity of the activities and reactions of man leads physicians trying to understand and classify them to talk about the mind and body as if they were distinct and separate entitites. The emphasis on specialization in medical practice of past decades increases this dichotomy of thinking.

But, in every area, including smoking and health, we are dealing with a whole entity; an interrelated complex of underlying, constitutionally determined, physical and psychic patterns of behavior.

Since causes of reaction are complex, so the causes of disease are multiple. The 1964 Surgeon General's Advisory Committee on smoking and health recognized this basic proposition: "All members," the Report notes, "shared a common conception of the multiple etiology of biological processes" and "all were thoroughly aware of the fact that there are series of events in occurrences and developments in these fields, and that the end results are the net effect of many actions and counteractions."

What we cannot tell from simple statistical association--even if it is assumed that the association is real--is whether both smoking and the various diseases statistically associated with it are correlated with other hidden factors such as psychological and physiological differences which themselves exert the true causal force. Accumulated data suggest a number of reasons to believe this is so. In a presidential address to the American Cancer Society 20 years ago, Dr. Eugene Pendergass stated about cancer: "There is a distinct possibility that within one's mind is a power capable of exerting forces which can either enhance or inhibit the process of this disease."

In a statement to the Congress in 1965, when the Congress was considering proposed warnings for cigarette packages and advertising, I warned that admonitory labeling of cigarettes could well fan the fire of youthful rebelliousness, rather than deter smoking by teenagers. At that time, it was long since well-known that public health officials had branded cigarettes smoking as deleterious to health.

As I predicted, surveys following the adoption of warning statements showed a rise in smoking by children, particularly girls, fast catching up with boys. Yet, a high percentage of youngsters believe that cigarette smoking can cause lung cancer, emphysema, heart disease. Relatively more smoking students in 1971 than 1964 believe that smoking impairs health. Still, it was reported that despite the anti-smoking campaigns and programs, approximately 1-1/4-1-1/2 million adolescents were starting smoking each year.

Education to the health hazards of smoking didn't deter them. Scare techniques and preaching didn't deter them. They smoke anyway. Teenagers rebel against restriction. Teenagers feel that they should be able to do the things they want to do when adults tell them they can't do them. Teenagers want to control what happens to them.

Consider teenage sex activity, which adolescents fully know can produce venereal disease, and pregnancy, with high rates of premature deliveries and congenital anomalies. Availability of contraceptive measures to teenagers, costly and extensive educational efforts about birth control, have not decreased the number of teenage pregnancies. These pregnancies have increased.

HR 4957 purports to find the present Federal, State and private initiatives insufficient to warn the American public about the dangers of smoking. This is not the fact. The fact is that awareness does not alter behavior after a point. The people reached by warnings about smoking have been reached. The others are not going to change their behavior, whatever the warning.

Different people smoke for different reasons, among them:

1) To cope with feelings of anxiety and stress;
2) For pleasurable relaxation;
3) To raise energy levels;
4) As a "suicidal act," not only consciously in the hope that health will be impaired, but also unconsciously as an inner-directing of aggressive behavior;
5) To control hostility and anger.

If we look at initiatives conveying health warnings that I have had a lot of experience with in the past fifty years--campaigns about alcohol, pill-popping, drugs, smoking, teenage pregnancies--we find that law (prohibition) did not abort drinking (30% or more of hospitalized patients today have alcohol-related problems); availability of contraceptives and birth control education to children did not decrease teenage pregnancies; numbers of people continue to smoke; there is more drug-taking throughout the adult and adolescent populations than ever before. The King County Medical Society Committee on Drug Abuse & Alcoholism, of which I am past Chairman, has addressed itself to campaigns for the education of physicians, as well as the general population. There has been no discernible change in behavior, unless it is in the direction of increasing tension-relieving behavior.

The way to learn about people's attitudes and reactions is to work closely, as I have, with thousands of patients. I have not seen a patient since the mid-fifties who was not aware of the assertion that they should not smoke. This information had even filtered down to children, who learned to tell their parents not to smoke in the belief that parental smoking is harmful to their parents and themselves.

But of course I see every day patients with cancer, and lung and heart disease, who have never smoked.

Medical "preventive" health is a personal problem. People are apprised of the dangers of smoking, but they have their own personal reasons for not giving up smoking. Dire medical advice coming from legislative bodies is not effective advice. Smoking is a personal medical matter, not a legal issue.

Stringent warnings have a negative effect. As The Smoking Digest, U.S. Dept. HEW, NCI, 1977 stated: "Fear-arousing messages actually increase smoking among the particularly vulnerable people and those with low self-esteem. Such messages tend to make the smoker defensive and harden his attitude and resistence. People who smoke to reduce anxiety also often smoke more after receiving a strong health threat in order to reduce their anxiety and fear."

If medicine is to teach "preventive" medicine, it must address itself to finding and promoting adaptive ways to deal with stress. The 1964 Surgeon General's report stated:

> "Stress seems to be related to smoking and there is evidence that the experience of stressful situations contributes to the beginning of the habit, to its continuation and to the numbers of cigarettes consumed."

The Report further stated:

> "Existence of an association between stress and tensions on the one hand and smoking behavior on the other can probably be accepted with a reasonable degree of confidence."

From the research material in the smoking field we could conclude that the same stress that stimulates heavy smoking may be the stress that precipitates the complex cancer process or produces the coronary-prone individual.

It is up to the medical profession to reduce stress in the population -- and not by fiats or mandates that engender guilt, anger and resistance, which in turn create more stress and can promote the very diseases they are trying to prevent.

It is costly and inappropriate for Congress to busy itself in this area. "Good health" and "good behavior" cannot be legislated. It is physicians who should and do address themselves to the person -- to the person's constitutional diathesis and self-motivation, and to techniques for reducing the person's stress with its deleterious physiological reactions and maladaptive responses.

CONCERNING THE "COMPREHENSIVE SMOKING PREVENTION ACT OF 1982"

G.N. Schrauzer, Ph.D.
La Jolla, California

I am Professor of Chemistry at the University of California, San Diego. I hold a Ph.D. degree in chemistry from the University of Munich and am the president and founder of the International Association of Bioinorganic Scientists. I am a member of several scientific societies, including the American Chemical Society, the Association of Clinical Scientists, and the American Public Health Association. I am the author of approximately 200 research publications and have edited 2 books.

My main research interests are in cancer-prevention, cancer epidemiology, trace minerals in human and animal nutrition, and various fields of experimental chemistry. I have done pioneering work on the prevention of cancer by the essential trace mineral selenium and in 1978 received a special award from the Santa Clara Section of the American Cancer Society.

As a chemist, cancer researcher and American Citizen I wish to comment upon the "Comprehensive Smoking Prevention Act of 1982" recently introduced into U.S. Congress. In this Act, it is stated, among other things, that "smoking is the number one cause of lung cancer in the United States". In my opinion, what role, if any, smoking plays in the causation of cancer, including lung cancer, has still to be determined.

Those who claim smoking causes cancer rely upon the reported statistical association and ignore the inconsistencies of the smoking causation theory in the scientific literature. For example, to date, no one has ever been able to produce lung cancer in laboratory animals through exposure to fresh, whole cigarette smoke. Moreover, the vast majority of smokers never develop lung cancer and there are serious inconsistencies in the epidemiological evidence and dose-response relationships. For example, a Japanese male smoking 50 cigarettes per day has a lower risk of dying from lung cancer than a British smoking doctor smoking only 1-14 cigarettes per day.

In addition, no ingredient or combination of ingredients, as found in tobacco smoke, has been shown to cause human lung cancer. Tobacco smoke is a very complex mixture of thousands of ingredients. Some of these ingredients, in isolation, have been reported to be carcinogenic in test animals. However, this does not mean that tobacco smoke is harmful to humans. Compounds that are carcinogenic in test animals, when applied in isolation, have been found to be anti-carcinogenic when applied in combination with each other. Since it is probable that the many hundreds of compounds present in smoke interact with each other, it is highly artificial to focus upon the effects of any one ingredient or combination of ingredients in isolation from the others. It has long been known that certain smoke constituents act as anti-carcinogens in test animals. For example, tobacco belongs to the selenium accumulating group of plants and selenium has been shown to possess anti-carcinogenic properties. Also, constituents of cigarette smoke previously thought to be lacking altogether in carcinogenic activity have recently been found to be anti-carcinogenic when applied with true carcinogens in test animals.

Cancer is an extremely complex, multifactorial disease. Studies indicate that many factors, other than smoking, are statistically associated with cancer. Some of these factors are familial predisposition, exposure to tumor viruses and other biological causing agents, exposure to ionizing radiation and industrial carcinogens, diet, exogenous environmental factors and stress.

Further, studies indicate that a number of agents normally present in foods may have activating effects on tumor viruses and that there is an association between lung cancer mortalities and other variables of life-style and diet. For example, the lung cancer mortalities are correlated with the per capita intakes of sugar and milk, and with the consumption of seafoods. It also has been repeatedly suggested and recently reaffirmed that the consumption of diets rich in pro-vitamin A (carotene) may have lung cancer protecting effects.

The "findings" in the "Comprehensive Smoking Prevention Act" have not been proven. Moreover, passage of the Act will divert attention from other etiologic leads to the disvantage of the American Public and the progress of the health sciences.

STATEMENT OF DR. CARL C. SELTZER

I am Dr. Carl C. Seltzer, Honorary Research Associate at the Peabody Museum, Harvard University and Professor of Nutrition, Tufts University. I was formerly Senior Research Associate at the Harvard University School of Public Health. My work in the smoking and health area is extensive and I have published over thirty-five (35) articles since 1964 on the subject, many dealing with the relationship between smoking and coronary heart disease (CHD). I was a consultant to the Surgeon General's Advisory Committee on Smoking and Health (contributed a section to the 1964 Report) and am presently a Fellow of the Council on Epidemiology of the American Heart Association. My investigative work in the heart disease field includes participation in studies involving the Framingham Heart Study, The American Medical Association, the Veterans Administration and the Kaiser-Permanente Foundation.

H.R. 4957 proposes that the following label statement appear on cigarette packages: "Warning: Cigarette smoking is a major cause of Heart Disease." This proposal is apparently based on the claim, put forth as a Congressional "finding" in the first part of the bill, that one third of deaths from CHD are "attributable to smoking."

These bald statements are not based on consistent, valid, demonstrable scientific evidence, and are without established proof. Apparently, they are based on the views of a succession of Surgeon Generals subsequent to the 1964 Report, on the statements of the American Heart Association, the Framingham Heart Study and others.

The primary basis for these views can best be characterized by statements from the 1981 Surgeon General's Report and by the Framingham Heart Study. The 1981 Report stated: "The effect of smoking on CHD risk fulfills many epidemiological criteria for a causal association: powerful, independent, dose related, and reversible." Kannel of the Framingham Heart Study stated: "Causal inferences are supported by the fact that the relationship is strong, consistent, demonstrated prospectively, independent of associated risk factors, and can be explained by the known effects of cigarette smoking on the cardiovascular apparatus. Finally, it has been shown that those who quit smoking have only half the risk of those who continue to smoke. Epidemiologic data have . . . tended to indicate an independent, transient, noncumulative, reversible, triggering effect of cigarette smoking." (JAMA, 1981)

Accordingly, the main evidence advanced for a causal relationship between cigarette smoking and CHD is essentially epidemiologic, to the effect that the association is (1) strong, (2) consistent, (3) dose-related, (4) independent of associated risk factors, and (5) reversible by stopping smoking. (6) In addition, the association is said to be explained by known effects of cigarette smoking on the cardiovascular system. Let us examine these claims in turn.

Claim 1. Is the relationship strong? The 1964 Surgeon General's Report stated that in the U.S. the mortality ratio of smokers to nonsmokers is 1.7. But, this is not a strong relationship, according to Professor Hutchison of the Harvard School of Public Health. (Bull NY

Acad Med, 1968) This independent observer feels that a mortality ratio of two or less is actually indicative of a weak relationship which may possibly be explained by confounding factors.

Can the relationship be characterized as "strong" when the 1979 Surgeon General's Report, although discussing smoking and health studies throughout the world, limits its conclusion to "men and women in the United States," and Kannel of the Framingham Study talks about the excess CHD death rates of smokers "in most of Western societies"? (AHJ, 1981) If the relationship between smokers and CHD is "strong," why is it not present in all Western societies or in non-Western societies?

Claim 2. Is the relationship consistent? Since no significant statistical associations between smoking and CHD have been reported in Finland, the Netherlands, Italy, Greece, Yugoslavia, Japan and Puerto Rico, it is clearly incorrect to claim that the relationship is consistent.

The relationship is not even consistent in the Framingham Study. It appeared to be consistent in initial analyses, but when standard risk factors and such psychological factors as Type A personality were controlled, it was found that "cigarettes smoked per day" were not predictive of CHD and myocardial infarction incidence in men, or of CHD and angina pectoris incidence in women. (Am J Epidemiol, 1980)

Can the relationship be characterized as consistent when the 1979 Surgeon General's Report concluded that "the predictive risk factor association of smoking with the incidence of angina pectoris is not clear"? The Framingham Heart Study also reports that "the relationship to angina pectoris is modest, if it exists at all." (JAMA, 1981)

Is the relationship consistent when the relative risk progressively declines with increasing age? Seltzer demonstrated and Kannel noted that the benefits of quitting smoking do not extend beyond the age of 65 as regards heart attacks. (Am J Med Sci, 1978; JAMA, 1981) Furthermore, the 20 year follow-up data of the Framingham study show no excess incidence of CHD in men or women 55 years and over. (Framingham data made available to Seltzer as consultant.)

Can the relationship be characterized as consistent when the Framingham Heart Study reports an absence of a relationship of smoking and CHD in women?

Claim 3. Is the relationship dose-related? CHD rates do not consistently show a rising gradient in relation to an increased amount of cigarette smoking. (JAMA, 1968) In some studies, the gradient is actually reversed. (JAMA, 1968) I found that data from the Framingham Study do not show a consistent rise in CHD risk with increasing amounts of cigarette smoking because of the "heterogenous" character of its "none" category, a deceptive procedure. The "none" category in Framingham consists of a combination of never smokers, ex-smokers, pipe and cigar smokers. A Framingham Study report confirmed that "there is no distinct dose-response relation [of CHD and death rates] with increasing amounts of cigarette smoking" over an 18-year surveillance period. (*Lancet*, 1974)

There is no significant dose-response relationship between smoking and CHD in studies of Finland, the Netherlands, Greece, Italy, Yugoslavia and Japan. (*Circulation*, 1970)

No claims have been made lately about the alleged association of CHD with duration of cigarette smoking. In the combined Albany and Framingham Heart Studies (*NEJM*, 1962), no association was found between duration of heavy cigarette smoking and risk of myocardial infarction. In a study of Canadian veterans, no consistent gradient occurred between CHD mortality and years of cigarette smoking. (*JAMA*, 1968) In Kahn's analysis of Dorn's U.S. veterans data, there was also no significant relationship between duration of cigarette smoking and risk of death from CHD. (*JAMA*, 1968)

Claim 4. Is the relationship independent of associated risk factors? Smoking has been found to be associated with CHD independently of associated risk factors in some studies when only cholesterol and blood pressure were considered. But this generally has not been the case when other risk factors were investigated, such as Type A personality, and other psychological factors (as seen above in the Framingham Study).

Claim 5. Does stopping smoking reduce the risk of CHD? This claim is based on studies from some Western countries which report that CHD rates of ex-smokers are lower than those of continuing smokers. The 1974 Lancet article on the Framingham study is the most widely quoted source for claiming that the rate of CHD among those who stop smoking is reduced to one-half that of continuing smokers. When I was provided with the CHD rates of Framingham "never smokers" (which had been omitted in the article), however, I was able to show that the CHD rates of ex-smokers were far below those of "never" smokers," while the rates of "never smokers" were not far below those of continuing smokers. (Seltzer letter to Lancet, 1977)

Additional evidence about the fallacy of the claim that stopping smoking reduces the rate of CHD comes from two other sources. The first is a study challenging the assumption by epidemiologists that ex-smokers and continuing smokers are alike in all pertinent characteristics, except for their smoking habits. If this assumption is false, which I believe it to be, then comparisons of the two groups would be biased as to their eventual CHD rates. Friedman, Seltzer, and others have shown that ex-smokers, when they still smoked, were not a representative sample of the population of continuing smokers. The data showed, in fact, that ex-smokers were at lower heart disease risk (and healthier) before they stopped smoking, than those persons who continued to smoke. (J Chron Dis, 1979) This would appear to account for the fact that ex-smokers have lower CHD rate outcomes than continuing smokers, not because they stopped smoking but because they are different kinds of persons to begin with through self-selection.

In a subsequent article published in the NEJM, Friedman and others tried to negate the results of that paper by applying theoretical correction techniques through multivariate analysis to the lower CHD-related characteristics of the ex-smokers. They claimed that the lower CHD-related characteristics actually made little difference, that ex-smokers still showed lower CHD outcomes than continuing smokers. Although this paper was criticized by Burch and Seltzer (Burch and Seltzer letters to NEJM, 1981), Friedman evaded virtually every criticism in his reply. (Friedman letter to NEJM, 1981) Friedman did not explain how he could accept the value of CHD-related characteristics (risk factors) in one paper and deny their importance in a second paper. Nor did Friedman explain why the mortality data in his later paper showed that never smokers exhibited higher CHD rates than the ex-smokers (the same anomaly as that found in the Framingham Study). Does this mean that it is better to begin smoking and then to quit than never to have smoked at all?

The second source of evidence which points to the fallacy of the claim that stopping smoking reduces the risk of CHD is the first large-scale "intervention" trial of the effect of smoking cessation in middle-aged London civil servants at high risk of cardiovascular disease. (J Epidemiol Comm Health, 1978) After nearly eight years of surveillance, the group subject to intervention for smoking cessation showed no improvement in its rate of mortality over the group which was not urged to stop smoking. Thus, the alleged reversibility of the risk to the smoker's life was not demonstrated.

This result is consistent with a more recent intervention trial from Norway which examined the effects of both smoking and cholesterol reduction. (Lancet, 1981) Although reductions in dietary cholesterol produced a statisticially significant decrease in CHD, reductions in smoking did not. This led the authors to conclude that past studies may have "overestimated the decline in risk of CHD" following reduced cigarette consumption.

Claim 6. Kannel of the Framingham Study claims that: "causal inferences . . . can be explained by the known effects of cigarette smoking on the cardiovascular apparatus." (JAMA, 1981) There is no satisfactory evidence for this claim, and, in fact, there are contradictions. The cold reality is that the mechanisms by which tobacco smoking allegedly causes CHD have not been established. Much of the evidence to support this statement is contained in Seltzer's article, "Smoking and Coronary Heart Disease: What Are We to Believe," published in the American Heart Journal as an editorial in September, 1980. This editorial discusses those statements of the 1979 Surgeon General's Report which illustrate the absence of firm evidence of mechanisms by which smoking affects CHD, such as: (1) little is known about the mechanisms by which smoking enhances atherogenesis, (2) further research is needed to show the mechanisms of sudden death and its precursor states, (3) the data on the effect of smoking on blood lipids are not very uniform, (4) the association between cholesterol and smoking is minimized, (5) the acute and transient effects of smoking are to increase heart rate and blood pressure to a minor degree, (6) smoking is not a risk factor for hypertension, (7) the association of smoking with necropsy manifestations of atherosclerosis, shown principally by Strong's group in New Orleans, now has been contradicted by a study by Holme, Strong and others who found that smoking did not show a significant association with coronary artery lesions. (Arteriosclerosis, 1981)

More evidence comes from other sources. An editorial in the August 1980 British Medical Journal states that the mechanism by which smoking affects coronary heart disease is unknown. The American Heart Association Heartbook states: "The mechanisms by which cigarette smoking is associated with higher rates of coronary heart disease are not yet fully understood." The 1981 Surgeon General's Report states: "Estimation of the impact of varying cigarettes on coronary heart disease risk is difficult, because the exact etiologic agent(s) have not been identified." Thus, the bill's proposal to label cigarette packages with levels of tar, nicotine, and carbon monoxide is not justified on scientific grounds. The Surgeon General admits that it has not been clearly demonstrated that these factors are prime etiologic agents in the causation of increased CHD in smokers.

This is also shown by the conclusion of the 1981 Report that despite radical changes in the composition of cigarettes over the last 10 to 15 years (filter, tar, nicotine), there was no clearly demonstrated effect on cardiovascular disease. This surprising result is clearly consistent with the proposition that amounts of tar, nicotine, and carbon monoxide in cigarette smoke have no demonstrable effects on CHD rates, and that these components of tobacco smoke are not proven etiologic agents.

Since most cardiologists believe that atherosclerosis is the most important factor in coronary heart disease development, they claim that smoking enhances atherosclerosis, despite the lack of definitive evidence. On the other hand, the Framingham Heart Study has disavowed the atherosclerotic argument and claimed that the effect of smoking on the heart is acute (not progressive), and disappears promptly when smoking is stopped. These are contradictory claims and illustrate the lack of definitive knowledge in this field.

It is clear from the above that extensive research data do not support the Congressional finding in H.R. 4957 that a major proportion of the CHD deaths in this country are attributable to smoking. In turn, the bill's proposed warning, "Cigarette smoking is a major cause of Heart Disease," is not scientifically valid.

In every instance, an examination of the claims made against cigarette smoking and CHD shows that they are either wrong, inconsistent, selective, unsubstantiated, or, in many respects, contrary to the statements of the Surgeon General, the American Heart Association and the Framingham Heart Study.

Relationships Between Family Smoking Habits, Individual Differences in Personality, and the Smoking Behavior of College Students

By

Charles D. Spielberger, Ph.D.
Professor of Psychology
Director, Center for Research in
Community Psychology
University of South Florida
Tampa, Florida 33620

I am Charles D. Spielberger, Professor of Psychology and Director of the Center for Research in Community Psychology at the University of South Florida in Tampa. I obtained my Ph.D. in Psychology at the University of Iowa in 1954, and have been a member of the psychology faculty at Duke University, Vanderbilt University and Florida State University, and directed the Doctoral Programs in Clinical Psychology at Florida State and the University of South Florida. I was also employed by the U.S. Public Health Service as a training specialist in psychology. I am certified in Clinical Psychology by the American Board of Psychologists and presently serve as Associate Editor for three major journals and on the editorial boards for a number of additional journals. I have contributed to the research literature on personality for more than 25 years, and have been interested in the association of personality and smoking behavior for the past five years.

Due to a long-standing commitment to host the Annual Meeting of the Society for Personality Assessment, in Tampa, Florida, March 11-13, 1982, in my capacity as official host for the meeting and member of the Society's National Board, I will be unable to appear in person before Congressman Waxman's Subcommittee to present the results of several recent studies in which I have been engaged, and which I believe are relevant to the Subcommittee's consideration of H.R. 4957. I have, therefore, prepared this statement for submission to the Subcommittee.

Over the past four years, I have conducted research on the relationships between family smoking habits, selected personality traits and the smoking behavior of college students. More than 1500 students have been interviewed or tested in this research. A Smoking Behavior Questionnaire (SBQ) was developed to elicit specific information about students' smoking behavior and the smoking habits of their families, and standardized personality inventories were administered to most of the participants in these studies.

The major findings that have emerged in this research are summarized in this paper. My research results are generally consistent with the increasing evidence that peer-group pressures are the single most important influence in the initiation of smoking. In this regard, it is noteworthy that the results of a related study which I am presently completing indicates that media advertising is consistently ranked near the bottom of a listing of ten factors which might be thought to influence college students to start smoking. This finding is strengthened because it was true for both females and males. (Please see Table 1, page 8.)

The second area of research reported on below provides evidence that smokers and non-smokers differ in personality, and thereby supports the hypothesis that constitutional-genetic factors contribute to the maintenance of smoking behavior.

My research results are set forth below in two sections. Relationships between family smoking habits and the initiation and maintenance of smoking behavior are reviewed in the first section. Next, relationships between important personality traits and smoking behavior are examined. Manuscripts for two papers that provide more detailed information about the research findings are included as an Appendix to this report. Both papers have been accepted for publication in scientific journals.

Family Smoking Habits and Student Smoking Behavior

For both males and females, students whose older brothers or sisters smoked were more likely to be smokers than those whose older siblings did not smoke. Older sisters appeared to have greater impact on the smoking behavior of their younger sisters than their younger brothers, whereas older brothers seem to have a similar influence on the smoking behavior of younger siblings of both sexes. These results are generally consistent with mounting evidence that peer group pressures are perhaps the single most important influence in the initiation of smoking.

A positive association was also found between the smoking behavior of college students and the smoking habits of their parents. If one or both parents smoked, their sons and daughters were more likely to be smokers than if neither parent smoked. These findings were generally consistent with results reported in eight previous investigations.

When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings appeared to have a stronger influence on the smoking behavior of younger siblings than their parents. Students whose older siblings were smokers were more likely to take up smoking than those whose older siblings were non-smokers, and parental smoking habits seemed to have no added influence on the smoking behavior of these students. Students with no older siblings, or whose older siblings were non-smokers, were less likely to be smokers themselves, but these students were more likely to take up smoking if one or both parents smoked than if neither parent smoked.

No differences were found in the smoking habits of the parents of current, occasional and ex-smokers, nor in the smoking habits of their older siblings. Thus, there was little evidence that family smoking habits influenced the maintenance of smoking behavior after the students had begun to smoke. Taken as a whole, the results indicate that family members influence students to take up smoking, but have little or no impact on the maintenance of smoking behavior.

The finding that the smoking behavior of students was more strongly associated with the smoking habits of older siblings than with parental smoking habits suggests that peer groups and other environmental influences are more important than constitutional-genetic factors in the initiation of smoking. However, the data on the relationship between personality and smoking that are discussed in the following section provide evidence that Smokers and Non-Smokers differ in personality, and thus support Eysenck's (1980) hypothesis that constitutional-genetic factors contribute to the maintenance of smoking behavior.

Personality and Smoking Behavior

Students classified as "Smokers" (Current, Occasional and Ex-Smokers) had significantly higher mean scores than Non-Smokers on the Extraversion, Neuroticism and Psychoticism scales of the Eysenck Personality Questionnaire (EPQ), and significantly lower scores on the EPQ Lie Scale. Although the observed differences between Smokers and Non-Smokers were similar for both sexes, women scored higher than men on the EPQ Neuroticism and Lie scales, and lower on Psychoticism. The differences between Smokers and Non-Smokers on all of the *EPQ* measures were also stronger for the female students than for the males.

Female Smokers had higher scores than Non-Smokers on the Trait Anxiety scale of the *State-Trait Personality Inventory* (*STPI*), whereas male Smokers had slightly lower anxiety scores than Non-Smokers on this scale. No significant differences were found between Smokers and Non-Smokers on the *STPI* Trait Curiosity and Trait Anger scales.

In evaluating the relationship between personality traits and the maintenance of smoking behavior, Ex-Smokers were found to have significantly higher *STPI* Curiosity scores than Current or Occasional Smokers, and female Current Smokers had *lower* *EPQ* Neuroticism scores and *lower* *STPI* Trait Anxiety scores than female Occasional and Ex-Smokers. The finding that female Current Smokers scored lower in both neuroticism and anxiety than female Occasional and Ex-Smokers was surprising, and suggested that smoking may serve primarily as a tension reducer for females who are generally higher in neuroticism than males.

Given the fact that Ex-Smokers had significantly higher Trait Curiosity scores than Current or Occasional Smokers, we may speculate that curiosity motivates Ex-Smokers to take up smoking, but this motive would be unlikely to stimulate them to continue to smoke. The finding must be replicated, however, in order to have confidence in this speculative interpretation.

While the finding that Smokers scored higher than Non-Smokers on all of the *EPQ* personality dimensions was consistent with results previously reported by other investigators, this was the first study to demonstrate a significant relationship between the EPQ measures and smoking behavior with an American sample. An article describing the relations between personality and smoking behaivor has been accepted for publication in the Journal of Personality Assessment. A preprint of this article is included in the Appendix.

Taken as a whole, the findings of our research provide additional support for Smith's (1970) and Matarazzo and Matarazzo's (1965) conclusions that smokers are more extraverted, neurotic and tense, and have stronger antisocial tendencies than non-smokers. The results also suggest that females, who are higher in neuroticism and trait anxiety than males, may take up smoking in order to reduce tension (Eysenck, 1980). The finding that female Current Smokers scored lower on neuroticism and trait curiosity than Occasional and Ex-Smokers further suggested that smoking may be an effective tension reducer for females who smoke regularly.

Conclusion

The research on family smoking habits reviewed above is consistent with existing evidence that peer pressures are the single most important influence in the initiation of smoking. The research on personality and smoking suggests that constitutional-genetic factors associated with personality differences contribute to the maintenance of smoking behavior.

March 8, 1982

Charles D. Spielberger
Charles D. Spielberger

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2. Matarazzo, J.D. and Matarazzo, R.G. Smoking. In: D. L. Sills et al. (Eds.), International Encyclopedia of the Social Sciences. New York: MacMillan, 1965.
3. Smith, G. M. Personality and smoking: A review of the empirical literature. In: W. A. Hunt (Ed.), Learning mechanisms and smoking. Chicago: Aldine, 1970.

Table 1

Rank Order, Means and Standard Deviations of Factors Reported by Male and Female College Students to Influence Them to Begin Smoking

Reason for Beginning to Smoke	Females (N=2[illegible])		Males (N=130)	
	Rank	$\bar{X}$ SD	Rank	$\bar{X}$ SD
See if I would enjoy it	1	2.61 0.95	1	2.52 0.92
Most friends smoke	2	2.52 1.14	3	2.41 1.16
Try something new	3	2.44 0.97	4	2.32 0.92
Thought it was satisfying because other people smoke	4	2.37 0.98	2	2.48 0.98
Made me feel more relaxed in social situations	5	2.14 1.05	5	2.02 1.08
Parents seemed to enjoy smoking	6	1.58 0.89	7	1.53 0.85
Did not want to refuse friends	7	1.51 0.89	6	1.55 0.87
Older siblings enjoyed smoking	8	1.48 0.83	9	1.46 0.83
Media advertisements	9	1.46 0.73	8	1.48 0.74
Parents disapproved - show independence	10	1.32 0.73	10	1.31 0.69

APPENDIX

Scientific Papers

1. Spielberger, C.D., Jacobs, G.A., Crane, R.S. & Russell, S.F. On the relation between family smoking habits and the smoking behavior of college students. International Review of Applied Psychology, In press, 1982.
2. Spielberger, C.D. & Jacobs, G.A. Personality and smoking behavior. Journal of Personality Assessment, In press, 1982.

International Review of Applied Psychology, in press.

On the Relation Between Family Smoking Habits and the Smoking Behavior of College Students

Charles D. Spielberger, [illegible] Jacobs, Rosario S. Crane
and Stephen F. Russell

University of South Florida

Running Head: Family Smoking Habits and Student Smoking Behavior

Abstract

This study investigated the relationship between the smoking behavior of college students (603 females; 352 males) and the smoking habits of their parents and older siblings. Students responded to a *Smoking Behavior Questionnaire* that required them to report whether they were Current Smokers, Occasional Smokers, Ex-Smokers, or Non-Smokers; similar information was obtained about their parents and older siblings. Of the females, 49% were classified as Smokers as compared to only 37% of the males. Students whose parents or older siblings smoked were more likely to be Smokers themselves; older siblings appeared to have a greater influence on the smoking behavior of younger siblings than did their parents. No differences were found in the smoking habits of the parents or the older siblings of Current, Occasional and Ex-Smokers. Contrary to previous investigations, there was no evidence of same-sex parental modeling of smoking behavior.

On the Relation Between Family Smoking Habits and the Smoking Behavior of College Students

The controversy on smoking and health has stimulated extensive interest in identifying factors that influence the initiation and maintenance of smoking behavior. In reviews of research in this field (Matarazzo & Matarazzo, 1968; Wohlford & Giammona, 1969; Evans, Henderson, Hill, & Raines, 1979; Leventhal & Cleary, 1977, 1980), social influence variables such as parental smoking habits and peer-group pressures have been repeatedly identified with the initiation of smoking, but relatively little is known about the factors that maintain the smoking habit.

Positive relationships between the smoking habits of parents and the smoking behavior of their children have been reported in eight studies (Banks, Bewley, Bl[illegible] Dean, & Pollard, 1978; Borland & Rudolph, 1975; Clausen, 1968; Horn, Courts, Taylor, & Solomon, 1959; Merki, Creswell, Stone, Huffman, & Newman, 1970; Palmer, 1970; Salber & MacMahon, 1961; Wohlford, 1970); only one study, which was based on a very small sample of college students, failed to find any relationship between these variables (Straits & Sechrest, 1963). Although an empirical relationship between parental smoking habits and children's smoking behavior seems firmly established, it is not clear whether this relationship reflects environmental or constitutional-genetic influences (see Eysenck, 1980).

Wohlford (1970) has called attention to the importance of examining the differential impact of the smoking habits of fathers and mothers on the smoking behavior of their sons and daughters. Sons were found to be more likely to smoke if their fathers smoked in four studies that examined this relationship

(Banks et al., 1978; Horn et al., 1959; Salber & MacMahon, 1961; Wohlford, 1970), and daughters were found to be more likely to smoke if their mothers smoked in three of these studies. Since no relationship was found between the smoking habits of fathers and daughters, nor between mothers and sons, same-sex parental modeling appears to have a stronger impact than genetic factors on children's smoking behavior.

Peer group pressure is also widely recognized as a primary factor in the initiation of smoking (e.g., Eysenck, 1980; Matarazzo & Matarazzo, 1968). Evidence of peer-group influence on the initiation of smoking was reported in six studies that investigated this relationship (Banks et al., 1978; Hill, 1971; Levitt & Edwards, 1970; Matthews, 197[illegible]; Merki et al., 1970; Palmer, 1970). Leventhal and Cleary (1980) have recently suggested that peers and parents are both important sources of environmental influence in cigarette smoking, and that older siblings may be even more important than other peers in influencing adolescents to initiate smoking. Consistent with this view, Banks et al. (1978) found that junior and senior high school students whose siblings smoked were more likely to be smokers themselves.

Only one published study could be located in which the relationship between family smoking habits and the maintenance of smoking behavior was investigated. Looye, Creswell and Stone (1972) found that secondary school students who were regular smokers were more likely to have parents and friends who smoked than students who had been regular smokers but subsequently stopped.

The goals of the present study were to investigate the influence of the smoking habits of parents and older siblings on the initiation and maintenance of smoking behavior for college students. On the basis of previous research findings, positive relationships were expected between family smoking habits and the initiation of smoking behavior, and between the smoking habits of fathers and sons, and mothers and daughters. The maintenance of smoking behavior was investigated by comparing ex-smokers with current and occasional smokers. On the basis of the findings reported by Lacye et al. (1972), current smokers were expected to be more likely than ex-smokers to have parents and older siblings who smoked.

Method

Subjects

The subjects were 955 undergraduate students enrolled in introductory level psychology courses at a large urban state university; the median age was 19 years. All students volunteered to take part in the study and received credit for their participation. The population served by the university is comprised primarily of families of low to average socio-economic status. More than 85 percent of the 24,000 students at the university are commuters, and many of them continue to reside with their parents.

The students were tested over a period of ten months. Sample I consisted of 460 students (166 males, 294 females) tested during the Winter and Spring quarters of the 1978-79 academic year; approximately 65 percent of the students enrolled in introductory psychology courses were included in this sample. Sample II consisted of 495 students (186 males, 309 females) tested during the Fall quarter of 1979; this sample included approximately 80 percent of the students enrolled in introductory psychology courses.

Construction of the Smoking Behavior Questionnaire (SBQ)

The SBQ is a 30-item self-report questionnaire designed to elicit specific information about students' smoking behavior and the smoking habits of their families. In constructing the SBQ, a number of questionnaires used to evaluate smoking habits in previous investigations were carefully reviewed (Clausen, 1968; Horn et al., 1959; Ikard, Green & Horn, 1969; Leventhal & Avis, 1976); items from these instruments were adapted for the present study.

A preliminary form of the SBQ was administered to 149 undergraduate students (52 males, 97 females) enrolled in introductory psychology courses. In responding to the questionnaire, the students were asked to indicate if they were interested in meeting in small groups to discuss reasons why college students start and continue to smoke. Each student was promised $2.00 for participating in these discussion sessions. A total of 81 students met with the investigators in small groups of 7 to 10 students. In order to permit in-depth discussion of the students' reasons for smoking or not smoking, there were separate groups for current smokers (N=27), ex-smokers (N=17), and non-smokers (N=37). The group discussions were audio tape-recorded.

On the basis of an analysis of the responses to the preliminary SBQ and a review of the audio tape, the final set of items for the form of the SBQ that was used in the present study were selected. The data presented in this study are based on the first part of the questionnaire, which inquired about the smoking habits of the students, their parents, and their older brothers and sisters.[1]

Procedures

The SBQ was administered as part of a larger test battery to groups of 20 to 100 students. In order to encourage participation in the study, most of the group-testing sessions were scheduled immediately after the psychology classes from which the students were recruited. At the beginning of each testing session, the students were required to read and then sign a Consent

Form, which informed them that participation in the study was entirely voluntary, and would consist of responding to several questionnaires. The students were then instructed to answer each questionnaire as honestly and accurately as possible. They were also informed that feedback sessions in which they could learn more about the study would be scheduled at the end of the term.[2] Prior to administering the SSQ, the following instructions were read to the subjects:

> Now, turn to the Cigarette Smoking Questionnaire...For Section 1, indicate whether you are a Current Smoker, Occasional Smoker, Ex-Smoker, or have never smoked by placing a check in the appropriate space under the column "Your Self". An "Occasional Smoker" is someone who smokes cigarettes from time to time but *not* everyday. Whatever your *own* smoking habits, be sure to also check whether your father, mother or older brother or sister is a Current, Occasional, or Ex-Smoker or has never smoked. (Students with more than one older sibling were asked to provide information about the older brother and/or sister who were the heaviest smokers.)

Results

In evaluating the relationship between family smoking habits and the *initiation* of smoking behavior, students classified as Current, Occasional and Ex-Smokers were considered "Smokers", and were treated as a single group in the statistical analyses. In examining the relation between the smoking habits of parents and older siblings and the *maintenance* of smoking behavior, the students who were classified as Current, Occasional, and Ex-Smokers were treated as separate independent groups. In all of the statistical analyses, students who reported they had never smoked were classified as "Non-Smokers".

Students who indicated they had experimented briefly with cigarettes, but had never become either regular or occasional smokers were also included in the Non-Smoker category.

The percentages of male and female students classified as Smokers or Non-Smokers in the two samples are reported in Table 1. Since these percentages were quite similar, the data for the two samples were combined. For the combined sample, the percentage of female smokers (49%) was substantially larger than the percentage of male smokers (37%); the difference between these percentages was statistically significant (X^2=12.94, df=1, $p<.001$).

Insert Table 1 about here

Family Smoking Habits and Students' Smoking Behavior

On the basis of the smoking habits of their parents, Smokers and Non-Smokers were assigned to one of the following three categories: (a) Neither parent smoked; (b) Mother or Father smoked, but not both; and (c) Both parents smoked. In Table 2, it can be noted that most students who reported that neither parent smoked were themselves Non-Smokers (females, 64%; males, 71%), and that the percentage of Smokers was higher if mother or father or both parents smoked than if neither parent smoked. In separate 3 x 2 Chi Square analyses, the relationship between parental smoking habits and the students' smoking behavior was statistically significant for females (X^2=11.03, df=2, $p<.01$), but not for males (X^2=3.37, df=2).

Insert Table 2 about here

In order to clarify the relationship between students' smoking behavior and the smoking habits of their parents, the percentages of female and male Smokers and Non-Smokers who reported that "Neither Parent Smoked" were compared in separate 2 x 2 Chi Square analyses with the percentages who reported that either mother or father smoked, or that both parents smoked. A significantly higher percentage of the females who reported that *mother or father smoked* (X^2=11.33, df=1, $p < .001$), or that *both parents smoked* (X^2=6.75, df=1, $p < .01$), were themselves Smokers. For males who reported that *mother or father smoked*, the percentage of Smokers (44%) was also significantly higher than for males (29%) who reported that *neither parent smoked* (X^2=3.98, df=1, $p < .05$), but not for males (35%) who reported that *both parents smoked* (X^2=0.86, df=1).

Surprisingly, the percentage of Smokers was slightly higher for students who reported that one parent smoked than for those who reported that both parents smoked, but these differences were not statistically significant for either females (X^2=1.17, df=1) or males (X^2=1.95, df=1). Since the trends for males and females in this comparison were similar, the data for the two sexes were combined and evaluated in a 2 x 2 Chi Square analysis, which approached significance (X^2=3.06, df=1, $p < .10$).

If there is same-sex modeling of parental smoking habits, it would be expected that the percentage of smokers would be greater among sons whose fathers smoked, and among daughters whose mothers smoked. Of the 336 females who reported that their mothers smoked, 52% were Smokers, as compared with 51% of the 414 females whose fathers smoked. For the 259 males whose fathers smoked, 38% were Smokers; 39% of the 188 males whose mothers smoked were themselves Smokers. Since the percentage of females and males who smoked was approximately the same, irrespective of whether their mothers or fathers smoked, there appears to be little evidence of same-sex modeling by children of the smoking behavior of their parents.

Of the 955 students who participated in the present study, 398 females and 223 males reported that they had older siblings, and 177 females and 80 males reported that they had both older brothers and older sisters. For the students with older siblings, it can be seen in Table 3 that the percentage of Smokers and Non-Smokers was approximately the same as for the total sample (compare column 1 in Table 3 with the data for the combined sample in Table 1).

Insert Table 3 about here

Students with older siblings were assigned to one of the following three categories: (a) Neither older brother nor older sister smoked; (b) Older brother or older sister smoked, but not both; (c) Older brother and older sister both smoked. The percentage of Smokers and Non-Smokers in each of these categories is reported in Table 3, in which it may be noted that the percentage of Smokers was highest for students with older brothers and sisters who smoked and lowest for those whose siblings were non-smokers. More than 70% of the students of both sexes whose older siblings were non-smokers were themselves Non-Smokers. Differences in the percentages of Smokers and Non-Smokers in the three categories were evaluated in 2 x 3 Chi Square analyses, and found to be significant for both females (X^2=43.39, df=2, $p<.001$) and males (X^2=9.96, df=2, $p<.01$).

The relationship between students' smoking behavior and the smoking habits of their older siblings was further evaluated in 2 x 2 Chi Square analyses. The percentage of Smokers was significantly higher for students whose older brothers or sisters smoked, and for students with both older brothers and sisters who smoked, than for students with older siblings who were non-smokers (Column 3 vs. Column 2: Females, X^2=30.85, df=1, $p<.001$; Males, X^2=7.86, df=1, $p<.01$; Column 4 vs. Column 2: Females, X^2=30.39, df=1, $p<.001$; Males, X^2=5.63, df=1, $p<.05$). Although the percentage of Smokers in the "brother and sister smoked" category was slightly higher than in the "brother or sister smoked" category, this difference was not statistically significant for either females (X^2=2.71, df=1) or males (X^2=0.46, df=1).

In order to evaluate the possibility that the smoking habits of older brothers and older sisters had differential influence on the smoking behavior of their younger siblings, the percentage of Smokers and Non-Smokers among students whose older siblings smoked was compared. For the 215 students whose older sisters smoked, the percentage of female Smokers (66%) was significantly higher than the percentage (46%) of male Smokers (X^2=8.12, df=1, $p<.01$), whereas the difference in the percentages of female (60%) and male (54%) Smokers for the 231 students whose older brothers smoked was not significant (X^2=0.73, df=1). Thus, older sisters seemed to have a greater impact on the smoking behavior of their younger sisters than on their younger brothers, whereas older brothers appeared to have a similar influence on the smoking behavior of younger siblings of both sexes.

In the preceeding analyses, older siblings appeared to have a greater influence on the smoking behavior of their younger siblings than did their parents. The combined influence of the smoking habits of parents and older siblings on the smoking behavior of the 955 students who participated in the present study is examined in Table 4. Evaluation of the differences in the percentages of Smokers and Non-Smokers for the six smoking categories defined by the smoking habits of parents and older siblings resulted in highly significant 2 x 6 Chi Squares for both females ($X^2=40.34$, df=5, $p < .001$) and males ($X^2=23.79$, df=5, $p < .001$). In general, the percentage of Smokers was higher for students with older siblings who were smokers (Columns 4-6), and lower for students whose older siblings (if any) were non-smokers (Columns 1-3).

Insert Table 4 about here

In order to further clarify the relationship between family smoking habits and the smoking behavior of the students in the present study, separate 2 x 3 Chi Squares were computed for students with older brothers and/or sisters who smoked (See Table 4, Columns 4-6), and for students with no older siblings who smoked (Columns 1-3). As previously noted, students whose older siblings smoked were more likely themselves to be Smokers, but parental smoking habits appeared to have no influence on their smoking behavior (females: $X^2=1.72$, df=[illegible]; males: $X^2=4.28$, df=2). In contrast, although students with no older siblings who smoked were less likely to be Smokers, these students were more likely to smoke if one or both parents smoked than if neither parent smoked (females: $X^2=15.53$, df=2, $p < .001$; males: $X^2=8.58$, df=2, $p < .01$).

Family Smoking Habits and the Maintenance of Smoking Behavior

In order to investigate the relationship between parental smoking habits and the maintenance of smoking behavior, students classified as Current, Occasional or Ex-Smokers were assigned, on the basis of the smoking habits of their parents, to one of the following three categories: (a) Neither parent smoked; (b) Mother or Father smoked, but not both; or (c) Both parents smoked. Surprisingly, as can be noted in Table 5, a higher percentage of females whose parents did *not* smoke were Current Smokers than was the case for students whose parents smoked. As expected, a larger percentage of the males were Current Smokers if one or both parents smoked than if neither parent smoked. However, when evaluated in 3 x 3 Chi Square analyses, these differences were not statistically significant for either females (X^2=5.72, df=4) or males (X^2=9.29, df=4).

Insert Table 5 about here

The relation between the smoking habits of older siblings and the maintenance of smoking behavior is presented in Table 6; these data are based on the 193 female and 88 male Smokers with older siblings. No significant differences were found in the percentages of female Current, Occasional and Ex-Smokers categorized on the basis of the smoking habits of their older siblings (X^2=3.07, df=4). Since the number of males with older brothers and sisters who smoked was very small, this group was combined with the males whose older brothers *or* sisters smoked. No significant differences were found in the percentages of Current, Occasional and Ex-Smokers for the combined group as compared with males who had neither older brothers nor sisters who smoked (X^2=4.20, df=2).[3]

Discussion

Of the 955 college students who participated in the present study, 49 percent of the females were classified as Smokers as compared to only 37 percent of the males. In commenting on recent trends in smoking behavior, Evans et al. (1979) observed: "The rate of smoking is *decreasing* among adult males, adult females, and male teenagers with the only *increase* -- a notable one -- occurring among females in their teens and early twenties" (p. 204). Thus, the finding in the present study that the percentage of smokers was significantly higher for females than for males appears to be consistent with recent trends in the smoking habits of young adults.

Positive relationships were found as predicted in the present study between the smoking behavior of students and the smoking habits of their parents. If one or both parents smoked, their sons and daughters were more likely to be smokers than if neither parent smoked. These findings were generally consistent with the results reported in six previous investigations (Banks et al., 1978; Borland & Rudolph, 1975; Horn et al., 1959; Merki et al., 1970; Palmer, 1970; Salber & MacMahon, 1961). Positive relationships have also been reported for females (Clausen, 1968) and for males (Wohlford, 1970) in two additional studies.

Positive relationships were also expected between the smoking habits of fathers and sons, and of mothers and daughters, but no evidence of same-sex parental modeling was found. A possible explanation is that the college students in the present study were older than the junior and senior high school students in previous studies (Horn et al., 1959; Salber & MacMahon, 1961; Banks et.al., 1970) for whom evidence of sex-linked modeling of smoking behavior was reported.[4] Thus, sex-linked parental modeling may influence some children to begin smoking at an earlier age, but this relationship may be attenuated or masked as the number of smokers increases during the high school and college years due primarily to peer group pressures. Consistent with this interpretation, the Surgeon General's report on Smoking and Health (1964) has suggested, "...that parents' influence affects the age at which children start smoking much more than it affects the ultimate taking or not taking up of the habit" (p. 370).

In the present study, students whose older brothers or sisters smoked were much more likely to be Smokers than those whose older siblings did not smoke (See Table 3). There was also some evidence that older sisters may have a greater impact on the smoking behavior of their younger sisters than their younger brothers, whereas older brothers appear to have a similar influence on the smoking behavior of younger siblings of both sexes. These results were generally consistent with Banks et al.'s (1978) finding that high school students with siblings who smoked were more likely to take up smoking than students whose siblings were non-smokers, and with the mounting evidence that peer group pressures are perhaps the single most important influence in the initiation of smoking (Eysenck, 1980; Levitt & Edwards, 1970; Matthews, 1974; Merki et al., 1970; Palmer, 1970).

When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings appeared to have a stronger influence on the smoking behavior of their younger siblings than did their parents. The students whose older siblings were Smokers were more likely to take up smoking than those whose older siblings were non-smokers, and parental smoking habits seemed to have no added influence on the smoking behavior of these students (See Table 4). Students with no older siblings, or with older siblings who were non-smokers, were less likely to be Smokers themselves, but these students were more likely to take up smoking if one or both parents smoked, than if neither parent smoked.

No differences were found in the smoking habits of the parents of Current, Occasional and Ex-Smokers in the present study, nor in the smoking habits of their older siblings. Thus, there was little evidence that family smoking habits influence the maintenance of the students' smoking behavior.

At the time the data for the present study were collected, the National Institute of Education (NIE, 1979) of the U.S. Department of Health, Education and Welfare initiated a major investigation of patterns of teenage smoking. In this study, a sample of 2639 American teenagers were interviewed by telephone to obtain information about their smoking habits and the smoking behavior of their families and friends. Although no statistical analyses were reported, the findings in the NIE study for high school students were similar in many respects to those obtained for college students in the present study. For example:

1. The data from the NIE study indicated that the percentage of girls who smoked was larger than the percentage of boys, whereas a decade earlier it was found that the percentage of boys who smoked was higher.

2. The NIE study found that if one or both parents smoked, their children were more likely to smoke, as was the case in the present study. There was also little evidence of same-sex modeling.

3. A positive relationship was found between the smoking behavior of teenagers and the smoking habits of their older siblings similar to the relationship for college students in the present study.

4. When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings had a stronger influence on their younger siblings than did their parents.

Taken as a whole, the results of the NIE study and the present investigation indicate that family members influence students to take up smoking. The fact that the smoking behavior of the students is more strongly associated with the smoking habits of their older siblings than with parental smoking habits suggests that environmental influences are more important than constitutional-genetic factors in the initiation of smoking. The findings in the present study that the percentage of smokers tended to be higher for students who reported that one parent smoked than for those who reported that both parents smoked provides further support for this interpretation.

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Footnotes

1. A copy of the SBQ may be obtained by writing to the first author.

2. Approximately two weeks before the end of the term, the students were notified of the dates and times for the feedback sessions and were encouraged to attend.

3. There was no evidence of a relationship between the combined influence of the smoking habits of parents and older siblings on the maintenance of smoking behavior of the students who participated in this study, as evaluated in 3 x 6 Chi Square analyses similar to those computed for Table 4.

4. Wohlford (1970) reported a positive relationship between the smoking habits of fathers and sons for college students, but not for mothers and daughters. It should be noted, however, that this relationship was based on a marginally significant correlation ($p < .06$) in "cigarette smoking frequency" for fathers and sons, rather than an evaluation of a correspondence in the prevalence of smoking behavior. Differences in Wohlford's findings and those of the present study might also reflect changes in the smoking patterns of young adults that have occurred over the past decade.

Table 1

Percentage of Male and Female Smokers and Non-Smokers in Two Samples of Undergraduate College Students

	Sample I	Sample II	Combined Sample
Total Number	460	495	955
Smokers	43%	46%	45%
Non-Smokers	57%	54%	55%
Females (N)	294	309	603
Smokers	48%	50%	49%
Non-Smokers	52%	50%	51%
Males (N)	166	[illegible]	352
Smokers	35%	[illegible]	37%
Non-Smokers	65%	51%	63%

Table 2

Relationship Between Parental Smoking Habits and the Smoking Behavior of College Students

	Number Of Subjects	Neither Parent Smoked	Mother Or Father Smoked	Both Parents Smoked
Females (N)	603	118	219	266
% Smokers	49%	36%	55%	50%
% Non-Smokers	51%	64%	45%	50%
Males (N)	352	66	124	162
% Smokers	37%	29%	44%	35%
% Non-Smokers	63%	71%	56%	65%

Table 3

Relationship Between the Smoking Behavior of College Students and the Smoking Habits of Their Older Siblings

	Students With Older Siblings	Neither Brother Nor Sister Smoked	Brother Or Sister Smoked	Brother And Sister Smoked
Females (N)	398	157	186	55
% Smokers	48%	29%	59%	71%
% Non-Smokers	52%	71%	41%	29%
Males (N)	223	94	109	20
% Smokers	39%	28%	47%	55%
% Non-Smokers	61%	72%	53%	45%

Table 4

Relationship Between the Smoking Habits of Parents and Older Siblings and the Smoking Behavior of College Students

	No Older Sibling Smoked[1]			Older Brother and/or Sister Smoked		
	Neither Parent Smoked	One Parent Smoked	Both Parents Smoked	Neither Parent Smoked	One Parent Smoked	Both Parents Smoked
Females (N)	79	124	160	40	94	106
% Smokers	22%	47%	46%	65%	65%	57%
% Non-Smokers	78%	53%	54%	35%	35%	43%
Males (N)	46	75	103	21	48	59
% Smokers	13%	37%	33%	62%	54%	39%
% Non-Smokers	87%	63%	67%	38%	46%	61%

[1] Students with no older siblings and those whose older siblings were non-smokers are included in this category.

Table 5

Relationship between Parental Smoking Habits and the Maintenance of Students' Smoking Behrvior

	Total Number Of Subjects	Neither Parent Smoked	Mother Or Father Smoked	Both Parents Smoked
Females (N)	295	43	119	133
% Current Smokers	40%	53%	34%	40%
% Occasional Smokers	26%	26%	28%	25%
% Ex-Smokers	34%	21%	38%	35%
Males (N)	130	19	54	57
% Current Smokers	31%	11%	26%	42%
% Occasional Smokers	29%	42%	35%	19%
% Ex-Smokers	40%	47%	39%	39%

Table 6

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Relationship Between the Smoking Habits of Older Siblings and the Maintenance of Students' Smoking Behavior

	Total Number Of Subjects	Neither Brother Nor Sister Smoked	Brother Or Sister Smoked	Brother And Sister Smoked
Females (N)	193	46	108	39
% Current Smokers	43%	37%	48%	36%
% Occasional Smoking	27%	33%	24%	28%
% Ex-Smokers	30%	30%	28%	36%
Males (N)	88	26	51	11
% Current Smokers	30%	39%	18%	64%
% Occasional Smokers	35%	42%	37%	9%
% Ex-Smokers	35%	19%	45%	27%

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PERSONALITY AND SMOKING BEHAVIOR

C. D. Spielberger and G. A. Jacobs
University of South Florida

Abstract

This study investigated the relationship between selected personality measures and the initiation and maintenance of smoking behavior. The Eysenck Personality Questionnaire (EPQ), the State-Trait Personality Inventory (STPI) and a Smoking Behavior Questionnaire were administered to 955 students (603 females; 352 males). Smokers had significantly higher scores than Non-Smokers on the EPQ Extraversion, Neuroticism and Psychoticism scales, and lower scores on the Lie Scale. Female Smokers had higher STPI Trait Anxiety scores than Non-Smokers, whereas male Smokers had lower anxiety scores than Non-Smokers. Female Current Smokers had lower Neuroticism and Trait Anxiety scores than Occasional and Ex-Smokers, and Ex-Smokers of both sexes had higher scores on the STPI Trait Curiosity scale than Current and Occasional Smokers. It was concluded that the initiation and maintenance of smoking behavior are influenced by different personality factors.

Personality and Smoking Behavior

The diverse literature on smoking and personality was reviewed by Smith (1970), who observed that smoking was positively associated with the following personality traits: extraversion (12 of 15 studies); antisocial tendencies (17 of 19 studies); and impulsive behavior (6 of 8 studies). Smith concluded that smokers were more extraverted and have more antisocial tendencies than non-smokers, and that the evidence is "reasonably convincing" that smokers are also more impulsive. Although there was some evidence that smoking was associated with neuroticism and anxiety, Smith considered these findings either inconsistent or based on too few studies to draw meaningful conclusions.

Matarazzo and Matarazzo have offered a somewhat different interpretation of the literature on smoking and personality. In their view: "The results, meager and poorly supported as they are, suggest the..... presence of a slightly higher number of 'extravert', and 'neurotic', and 'tense' individuals among the smokers as compared to the non-smokers" (1965, p. 377).

The studies published since Smith's (1970) review provide further evidence that smoking is associated with extraversion and antisocial tendencies (Brackenridge & Bloch, 1972; Kanekar & Dolke, 1970; Jacobs & Spilken, 1971). Smokers also have higher scores than non-smokers on Eysenck's Psychoticism Scale (Jamison, 1978; Powell, 1977; Powell, Stewart, & Grylls, 1979), which is assumed to measure impulsive, antisocial behavior (Eysenck & Eysenck, 1975). In addition, there is growing evidence that smokers are more tense, neurotic and anxious than non-smokers as inferred from higher scores on Eysenck's Neuroticism Scale (Brackenridge & Bloch, 1972; Gupta, Sethi, & Gupta, 1975; Powell et al., 1979) and the Taylor (1953) Manifest Anxiety Scale (Houston & Schneider, 1973; Schneider & Houston, 1970), a measure of trait anxiety (Spielberger, 1966, 1972).

There are two major difficulties in interpreting the research findings on smoking and personality. First, the definition of 'smokers' and 'non-smokers' has varied from study to study. A second problem is the failure to distinguish between factors that influence people to begin to smoke and that contribute to the maintenance of smoking behavior once it has been established. In evaluating the association between personality and the initiation of smoking, current smokers and ex-smokers should be classified as smokers. In examining the effects of personality variables on the maintenance of smoking behavior, current smokers and ex-smokers must obviously be considered as separate groups. It may also be important to distinguish occasional smokers (e.g., people who smoke from time to time but not every day) from regular smokers, ex-smokers and non-smokers.

The primary goal of the present study was to investigate relationships between extraversion, neuroticism and psychoticism as measured by the Eysenck Personality Questionnaire (EPQ) and the initiation and maintenance of smoking behavior for American college students. Although the scales developed by Eysenck and his associates have been widely used in smoking research in England, Australia and India, no studies could be found in which these scales were used to investigate relationships between personality and smoking with American subjects. A second goal of this study was to examine the association between smoking behavior and trait anxiety as measured by the State-Trait Personality Inventory (STPI, Spielberger, 1979).

On the basis of previous research findings, smokers were expected to score higher than non-smokers in trait anxiety, and on the EPQ Extraversion, Neuroticism and Psychoticism scales. Since sex differences have been reported in a number of previous studies of personality and smoking behavior (see Eysenck, 1980), these relationships will be separately evaluated for males and females.

Method

Subjects

The subjects were 955 undergraduate students (603 females, 352 males) enrolled in introductory level psychology courses at a large urban state university; the median age was 19 years. These students volunteered to take part in the study and received course credit for their participation. The sample consisted of more than 70 percent of the students who were enrolled in introductory psychology at the time the study was conducted.

Test Instruments

The Eysenck Personality Questionnaire (EPQ). The EPQ (Eysenck & Eysenck, 1975), the most recent form of a series of personality inventories developed over the past 30 years by Eysenck and his colleagues (Eysenck, 1952; Eysenck, 1959; Eysenck & Eysenck, 1968), consists of 90 true-false items. The EPQ yields scores on Extraversion (E), Neuroticism (N) and Psychoticism (P), the three dimensions of Eysenck's personality theory. It also includes a Lie (L) scale.

The State-Trait Personality Inventory (STPI). The STPI was developed to measure anxiety, curiosity and anger as emotional states and personality traits (Spielberger, 1979). This 60-item self-report inventory is similar in format and conception to the *State-Trait Anxiety Inventory*

(Spielberger, Gorsuch, & Lushene, 1970). The three 10-item STPI trait scales require the respondents to report the *frequency* that they have experienced the specified personality characteristics by rating themselves on the following four-point scale: 1) Almost never; 2) Sometimes; 3) Often; 4) Almost always. Persons with high STPI scores on a particular personality trait are more prone to experience the emotional states associated with that trait than persons with low scores. Although the primary interest in the present study was in trait anxiety, the STPI Trait Curiosity and Trait Anger scales were also administered for exploratory purposes.[1]

The Smoking Behavior Questionnaire (SBQ). The SBQ is a 50-item self-report questionnaire designed to elicit specific information about students' smoking behavior and the smoking habits of their families.[2] In constructing the SBQ, a number of questionnaires used to evaluate smoking habits in previous investigations were carefully reviewed (Clausen, 1968; Horn, Courts, Taylor, & Solomon, 1959; Ikard, Green & Horn, 1969; Leventhal & Avis, 1976), and some of the items from these instruments were adapted for the present study. Information about the construction of the SBQ and the relation between student smoking behavior and family smoking habits is reported by Spielberger, Jacobs, Crane, and Russell (in press).

Procedure

In order to encourage and facilitate participation in the study by a large proportion of the students enrolled in introductory psychology courses, the testing sessions were scheduled immediately after the classes from which the students were recruited. Groups of 20 to 100 students were tested over a 10-month period. At the beginning of each testing session, the students were informed that the purpose of the study was to "learn more about the feelings and attitudes of college students". They were also informed that feedback sessions to provide additional information about the research would be scheduled near the end of the term. The students then read and signed a Consent Form, advising them that participation in the study was entirely voluntary, and would consist of completing several questionnaires.

The test instruments were administered in the following order: (a) the STPI; (b) the SBQ; (c) the EPQ. The STPI and the EPQ were given with standard instructions. On the SBQ, which inquired about past and present cigarette smoking habits, students were asked to report whether they were Current Smokers, Occasional Smokers, Ex-Smokers or Non-Smokers. The instructions defined an "Occasional Smoker" as: "Someone who smokes cigarettes from time to time but not everyday." A "Non-Smoker" was defined as someone who had never smoked, or had only experimented briefly with cigarettes, but never became a regular or Occasional Smoker.

Results

The percentages of students who indicated that they were Current Smokers, Occasional Smokers, Ex-Smokers or Non-Smokers are reported in Table 1. Current Smokers, Occasional Smokers and Ex-Smokers were classified as Smokers in determining the percentage of female and male "Smokers" reported in Table 1.

Insert Table 1 about here

Personality and the Initiation of Smoking Behavior

In evaluating the association between the personality measures and the initiation of smoking, the scores of Smokers and Non-Smokers on the

EPQ and the STPI scales were compared. The means and standard deviations for each EPQ and STPI scale are reported in Table 2. These data were evaluated in separate 2 x 2 factorial analyses of variance in which Groups (Smokers vs. Non-Smokers) and Sex were the independent variables; the results of these analyses are also reported in Table 2.

Insert Table 2 about here

The significant Groups main effects for all four EPQ scales indicated that Smokers had higher scores than Non-Smokers on Extraversion, Neuroticism and Psychoticism, and lower Lie scale scores. The significant Sex main effects for the EPQ Neuroticism, Psychoticism and Lie scales reflected the fact that the females scored higher than the males on the Neuroticism and Lie scales, and lower on Psychoticism. None of the Groups by Sex interactions were significant for any of the EPQ scales.

While the differences on the EPQ scales were in the same direction for both sexes, these differences were larger in magnitude for the females, except for Neuroticism on which comparable differences were found. In the separate analyses for each sex, the female Smokers scored significantly higher than Non-Smokers on Psychoticism and Extraversion, and significantly lower on the EPQ Lie scale. Male Smokers also had significantly lower Lie scale scores than Non-Smokers.

For the STPI scales, the only statistically significant differences were the Sex main effect and the Groups by Sex interaction for trait anxiety. These findings reflected the fact that females had higher anxiety scores than males, and that female Smokers had higher anxiety scores than Non-Smokers, whereas male Smokers had lower anxiety scores than Non-Smokers. No significant differences were found for either sex in the separate analyses of the STPI scales.

Personality and the Maintenance of Smoking Behavior

The mean EPQ and STPI scores of the female and male Current, Occasional and Ex-Smokers are reported in Table 3. Associations between the maintenance of smoking behavior and each personality measure were evaluated in 3 x 2 factorial analyses of variance in which Groups (Current, Occasional and Ex-Smokers) and Sex were the independent variables. Summaries of these analyses are also reported in Table 3, along with the findings in the separate analyses of the data for female and male students.

Insert Table 3 about here

There were no statistically significant Group differences in the overall analyses of the EPQ scores, but sex main effects were found for the Neuroticism and Psychoticism measures. The females scored higher on N and lower on P than males, as was noted in the preceding analyses. Occasional Smokers of both sexes also had lower Lie scores than the Current and Ex-Smokers, who were quite similar on this scale. In the separate analyses for females, the Current Smokers had lower Neuroticism scores than the Occasional and Ex-Smokers.

In the analyses of the data for the STPI scales, the statistically significant Groups effect for Trait Curiosity reflected the finding that Ex-Smokers of both sexes had higher scores on this measure than Current and Occasional Smokers. A significant Sex main effect was also found for Trait Anxiety, which indicated that females had higher anxiety scores on this scale than males, as was noted in the preceding analyses. In the separate analyses for females, the significant Groups effect for the STPI trait anxiety measure reflected the fact that Current Smokers had lower anxiety scores than Occasional and Ex-Smokers.

Discussion

In the present study, Smokers scored higher than Non-Smokers on the EPQ Extraversion, Neuroticism and Psychoticism scales, and Non-Smokers had higher Lie scores than Smokers. While these findings were generally consistent with results previously reported by other investigators (Brackenridge & Bloch, 1972; Gupta et al., 1975; Powell et al., 1979), this is the first study to obtain such relationships for an American sample.

The differences observed between Smokers and Non-Smokers on the EPQ scales were in the same direction for both sexes, but these differences were larger in magnitude for the females. The findings in the present study that females scored higher than males on the Neuroticism and Lie scales, and lower on Psychoticism, were consistent with the results reported for British subjects in the EPQ Test Manual (Eysenck & Eysenck, 1975).

In evaluating the association between the STPI scales and the initiation of smoking, it was found that female Smokers had *higher* anxiety scores than Non-Smokers, whereas male Smokers had *lower* anxiety scores than Non-Smokers. The results for the females are consistent with findings reported by several previous investigators that smokers scored higher in anxiety than non-smokers (Houston & Schneider, 1973; Schneider & Houston, 1970), and support Matarazzo and Matarazzo's (1965) conclusion that there are, "...a slightly higher number of...'neurotic', and 'tense' individuals among smokers as compared to the non-smokers" (p. 377). The findings in the present study of a positive association between smoking and anxiety for females and a negative association for males points up the importance of taking the sex of the subject into account in an investigation of the relationship between smoking and personality.

In evaluating the association between personality and the maintenance of smoking behavior, no significant differences were found for Current, Occasional and Ex-Smokers in the overall analyses of the EPQ *E*, *N* and *P* scores. However, in the separate analyses for females, the Current Smokers had *lower* scores on Neuroticism than the Occasional and Ex-Smokers. A similar trend was also found for the males, but this difference was not statistically significant, perhaps due to the fact that the magnitude of the differences were larger for the females and the male sample was smaller. Female Current Smokers also scored lower on the STPI Trait Anxiety Scale than Occasional and Ex-Smokers.

Taken together, the results of the present study suggest that females, who are higher in neuroticism and trait anxiety than males, may take up smoking in order to reduce tension (Eysenck, 1980). The finding that female Current Smokers scored lower on neuroticism and trait curiosity than Occasional and Ex-Smokers further suggests that smoking may be an effective tension reducer for those females who smoke regularly. However, an alternative explanation is that occasional smokers and ex-smokers are higher in neuroticism and trait anxiety before they begin to smoke. A longitudinal prospective study of smoking behavior will be required to evaluate these alternative interpretations of the present findings.

An interesting new finding in the present study was that Ex-Smokers of both sexes had higher scores on the STPI Trait Curiosity Scale than Current and Occasional Smokers. We may speculate that curiosity is an important factor in motivating the Ex-Smoker to take up smoking, and that this motive would be unlikely to stimulate them to continue smoking. Thus, for Ex-Smokers, curiosity may influence the initiation of smoking, but not the maintenance of smoking behavior. The finding must be replicated, however, in order to have confidence in this speculative interpretation.

Smokers of both sexes had lower scores on the EPQ Lie Scale (see Table 2), and Occasional Smokers of both sexes had lower Lie scores than Current and Ex-Smokers (See Table 3). Although the Lie Scale was originally designed to measure the tendency to dissimulate ("fake good"), recent research suggests that low scores on this scale may be associated with non-conforming and rebellious attitudes (Eysenck, 1980). Therefore, the finding in the present study that Smokers had lower Lie scores was quite consistent with Smith's (1970) observation that smoking was associated with antisocial tendencies in 17 of 19 studies. The fact that Occasional Smokers had lower Lie scores than Current or Ex-Smokers further suggests that Occasional Smokers may take up smoking as a non-conformist behavior, and may also resist pressure from their peers to be regular smokers.

Of the 425 students classified as Smokers in the present study, 116 (27%) smoked only "from time to time, but not every day", and there were critical personality differences between these Occasional Smokers and Current Smokers. Moreover, the relationships between personality and the initiation and maintenance of smoking appeared to be somewhat different for men and women. Thus, it would seem important to distinguish between current and occasional smokers, and to take sex differences into account in future investigations of personality and smoking behavior.

Taken as a whole, the results of the present study provide additional support for Smith's (1970) and Matarazzo and Matarazzo's (1965) conclusions that smokers are more extraverted, neurotic and tense, and have stronger antisocial tendencies than non-smokers. Nevertheless, perhaps the main conclusions that can be drawn from this study are that the relation between smoking and personality is exceedingly complex, and that different personality factors may influence the initiation and maintenance of smoking behavior.

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Footnotes

1. The STPI state scales were also administered as a part of the test battery, but will not be considered here because the main concern in the present paper is with the association between individual personality traits and smoking behavior.

2. A copy of the SBQ may be obtained by writing to the first author.

Table 1

Number and Percentage of Current Smokers, Occasional Smokers, Ex-Smokers and Non-Smokers

	Females		Males	
	N	*%*	*N*	*%*
Non-Smokers	308	51%	222	63%
Smokers	295	49%	130	37%
Current Smokers	117	19%	40	11%
Occasional Smokers	78	13%	38	11%
Ex-Smokers	100	17%	52	15%
Total Sample	603	100%	352	100%

Table 2

Means, Standard Deviations, and Analyses of Variance of the Scores of Smokers and Non-Smokers on the Eysenck Personality Questionnaire (EPQ) and the Trait Scales of the State-Trait Personality Inventory (STPI)

Personality Measures	Groups X Sex ANOVA			Females			Males		
	F-Ratios								
	Groups	Sex	Int	Smokers (N=260)	Non-Smokers (N=274)	F-Ratio	Smokers (N=121)	Non-Smokers (N=207)	F-Ratio
EPQ									
Extraversion	4.50*	1.32	0.75	15.74 4.19	14.97 4.57	4.13*	15.10 4.18	14.81 4.42	0.34
Neuroticism	5.41*	32.00***	2.13	13.12 4.83	12.51 5.24	1.97	11.01 5.93	10.44 5.25	0.82
Psychoticism	4.97*	38.35***	1.52	3.48 2.36	2.79 2.19	12.19***	4.23 2.62	4.08 2.69	0.24
Lie Scale	12.81***	7.98**	1.44	6.45 3.47	7.48 3.83	10.67***	5.83 3.22	6.68 3.71	4.39*
STPI									
T-Anxiety	1.30	10.26***	4.90**	20.23 5.60	19.45 5.83	2.52	18.19 4.84	19.77 5.21	1.01
T-Anger	0.55	0.18	0.62	19.79 5.00	19.46 5.23	0.55	19.50 4.83	19.41 4.90	0.02
T-Curiosity	0.12	1.14	1.51	28.93 4.57	29.19 4.72	0.41	29.12 4.65	28.46 4.48	1.57

*p .05
**p .01
***p .001

Table 3

Means, Standard Deviations and Analyses of Variance of Scores on the Eysenck Personality Questionnaire and the State-Trait Personality Inventory for Female and Male Current Smokers, Occasional Smokers and Ex-Smokers

Personality Measures	Group X Sex ANOVA F-Ratios			Females				Males			
	Group	Sex	Int	Current Smokers (N=102)	Occas. Smokers (N=66)	Ex-Smokers (N=92)	F-Ratio	Current Smokers (N=38)	Occas. Smokers (N=34)	Ex-Smokers (N=49)	F-Ratio
EPQ											
Extraversion	1.89	1.56	0.58	16.1 3.9	15.6 4.7	15.5 4.1	0.58	15.9 4.0	15.4 4.0	14.3 4.3	1.73
Neuroticism	2.25	14.95***	0.48	12.2 5.0	13.5 4.3	13.9 4.9	3.56*	10.5 5.8	11.6 6.6	11.0 5.7	0.27
Psychoticism	1.47	8.38**	0.13	3.7 2.4	3.6 2.4	3.1 2.2	1.66	4.5 3.1	4.1 2.7	4.1 2.2	0.35
Lie	3.28*	2.57	0.12	6.6 3.4	5.7 3.4	6.9 3.5	2.49	6.1 3.1	5.2 3.1	6.1 3.4	0.82
STPI											
Trait Anxiety	2.32	13.28***	1.73	19.0 5.3	21.1 5.8	21.0 5.6	4.17*	18.4 4.8	18.5 4.9	17.9 4.9	0.20
Trait Anger	0.61	0.36	0.35	19.5 5.4	20.0 5.3	20.0 4.3	0.36	19.3 5.7	20.3 4.9	19.1 4.0	0.66
Trait Curiosity	4.20*	0.11	0.14	28.9 4.7	28.0 4.3	29.6 4.6	2.42	28.8 4.5	28.1 4.8	30.1 4.6	1.89

*p .05
**p .01
***p .001

March 8, 1982

Cigarette Smoking of Pregnant Women

A note regarding the "Comprehensive Smoking Prevention Education Act of 1982"

HR 5653

My name is Bea J. van den Berg. Currently I am director of the Child Health and Development Studies, a research unit of the School of Public Health of the University of California at Berkeley.

I received my medical doctor's degree in the Netherlands and in 1965 I joined the research staff of the Child Health and Development Studies that were designed and directed by the later Dr. J. Yerushalmy, professor of Biostatistics at the University of California at Berkeley. My CV is enclosed with the copy of my statement.

I am here to express my concern about the statements made in the proposed "Smoking Prevention Education Act of 1982" regarding an increased risk of spontaneous abortion, stillbirth, premature birth, child weight deficiencies and birth defects in pregnant women who smoke.

For many years I have been involved in research regarding pregnancy and pregnancy outcome, and research results do not support these statements.

Our studies were among the very first to identify the lower birth weight of babies born to smoking mothers as compared with that of non-smoking mothers. However, our studies did not indicate an increased risk of abortion and still births among smoking pregnant women nor are our studies supportive of the stated increased risk of birth defects.

For almost two decades, at the Child Health and Development Studies, we have studied the pregnancy outcome of women who smoked cigarettes during pregnancy, in comparison with women who never smoked or who stopped smoking before or early in their pregnancy. These studies were based on interviews, early in their pregnancies, of some 15,000 women who were members of Kaiser Foundation Health Plan, and who enrolled in the Child Health and Development Studies. Extensive information was obtained from the medical records of the mothers during their pregnancy and delivery and from the medical records of the children from birth to at least age five.

Our studies are prospective and observational; prospective because the smoking data were assembled before the outcome of the pregnancy was known. This approach avoids a possible recall bias that might occur when the mothers are interviewed after the termination of the pregnancy. An unfavorable pregnancy outcome might affect the mothers' recall of any event that occurred during her pregnancy, including smoking habits. Our studies are observational and not experimental; this signifies that not the researcher, but the women themselves decide whether or not to smoke cigarettes. The women in these separate groups might be different, not only in their smoking habits, but also in various other known or unknown characteristics. This methodological problem, coined by Dr. J. Yerushalmy as the problem of self-selection, has to be taken into consideration if we compare the pregnancy outcome of women who do smoke with the pregnancy outcome of women who do not smoke.

I would like to summarize the results of our studies to date that relate to the statements concerning pregnancy in the proposed Bill.

On birth weight. As early as in 1962 Dr. Yerushalmy reported an increased proportion of low birth weight infants among the offspring of smoking pregnant women as compared with the offspring of non-smoking pregnant women. The observed association may indicate a cause-and-effect relationship but it may also be influenced by differences between smokers and non-smokers in characteristics other than smoking. A later study indeed showed that a variety of other maternal characteristics, such as socioeconomic status, maternal age and ethnic background, influence the association between smoking during pregnancy and birthweight of offspring. Controlling for a number of these variables reduces the crude difference in the birthweight of the offspring of both groups of women.

Yerushalmy's later work on maternal smoking was aimed at developing methodologies and designing studies to test the causal hypothesis against the alternative hypothesis that increased incidence of low birth weight is due to the differences between smoking and non-smoking pregnant women. One such study evaluated the frequency of low birth weight among the offspring of mothers who began smoking after the birth of the infants. It was found that women who subsequently became smokers also had a high incidence of low birth weight babies during the period before they started to smoke. This finding cannot be explained by the causal hypothesis and underscores the need for a larger study to confirm or refute this finding.

On abortion and stillbirth. Our statistical study, comparing for each week of gestation the risk of fetal death for smoking and non-smoking pregnant women, failed to find a difference between the two groups. These results, based on the pregnancy outcome of 15,000 pregnant women, do not provide evidence that smoking during pregnancy raises the risk of spontaneous abortion and stillbirth.

On birth defects. We recently published the results of our evaluation of the incidence of congenital anomalies (or birth defects) among the 14,735 children born to women who never smoked, to women who were past smokers or to women who smoked during pregnancy. The children were offspring of mothers enrolled in the Child Health and Development Studies. Our data did not show a difference in the incidence of severe congenital anomalies among children of smoking women and children of women who never smoked. We also looked at the incidents of non-severe congenital anomalies. We found that, while the incidence of these non-severe birth defects among children of heavy smokers (more than 20 cigarettes per day) was slightly more than among children of non-smokers, the incidence among children our light smokers (less than 20 cigarettes per day) was equal to that of the children of non-smokers. These observations, again, do not support the statement in the proposed Bill that cigarette smoking by pregnant women may result in birth defects.

In conclusion, our data do not support the statements in the proposed Bill.